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Original Communications

CYSTOCELE AND HIGH RECTOCELE*

BY THOMAS J. WATKINS, M.D., CHICAGO, ILL.

IT IS not necessary to discuss in detail cystocele and high rectocele before this Society. Only some of the newer and more important observations will be considered.

CYSTOCELE

Certain features of cystocele have become so generally accepted that they have ceased to be subjects for serious discussion, namely, (1) that cystocele is hernia of the bladder; (2) that the lesion is essentially a rent in the vesico-vaginal floor; (3) that the operative treatment should comprise a thorough dissection of the hernia, a firm closure of the ring and restoration of the bladder and urethra to their normal positions.

Various opinions obtain as to the best method of closure of the ring, and reposition of the urethra and bladder. The points which I desire to stress will be presented largely by means of illustrations.

Fig. 1 represents the vaginal mucosa separated from the bladder and most of the fascia. The bladder has been separated from the cervix and the finger occupies the hernial canal. The extent and characteristics of the fascial rent can now be easily observed. *The tear is essentially transverse and not longitudinal.* Etiologically this could not be otherwise. The tension exerted upon the fascia which produces a bladder hernia results from the fetal head forcing down the cervix during labor and ceases only when the cervix retracts. Longitudinal tension produces a transverse lesion. It is impossible

*Read at the Forty-eighth Annual Meeting of the American Gynecological Society, Hot Springs, Va., May 21-23, 1923.

NOTE: The Editor accepts no responsibility for the views and statements of authors as published in their "Original Communications."

to visualize any extensive lateral tension upon the fascia during labor. The urethral displacement that always obtains in cases of cystocele can only result from a transverse tear. The next illustration (Fig. 2) is from Frank's work and depicts the vesicovaginal fascia as viewed from above. The dotted lines we believe show essentially the nature of the lesion in cases of cystocele. The tension upon

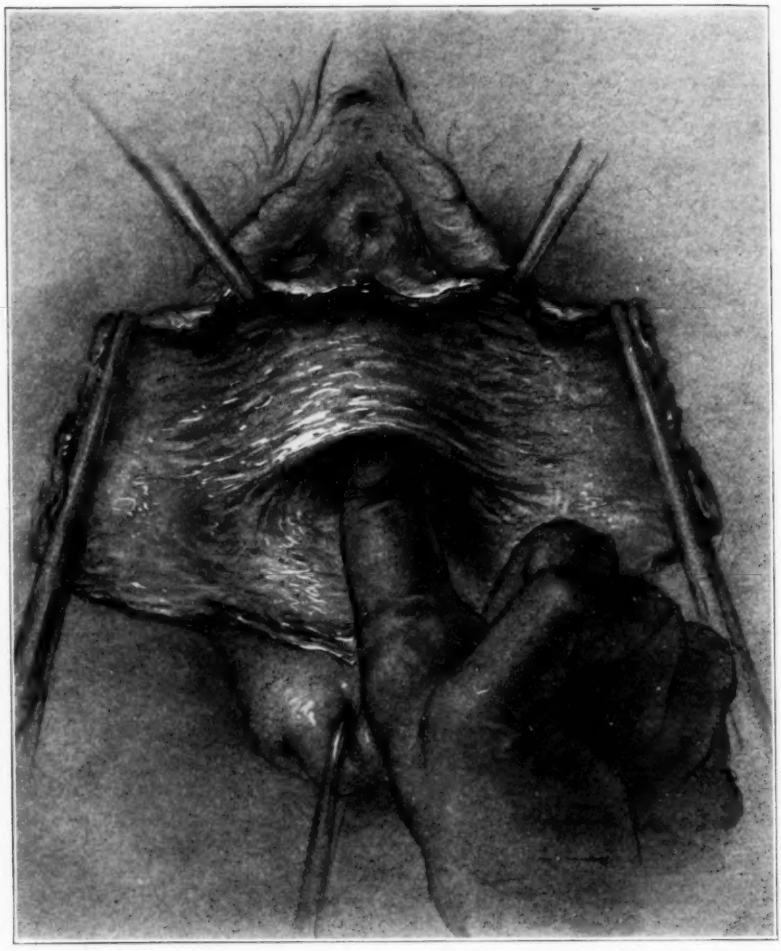

Fig. 1.—Vaginal mucosa separated from bladder and most of fascia. Finger occupies hernial ring.

the fascia during labor has separated it from the cervix and the base of the broad ligament. Fig. 3 is a diagrammatical illustration of the nature of the fascial lesion viewed from above.

Closure of the Hernial Ring.—This should accomplish (1) cure of the hernia and (2) cure of the urethrocele (urinary incontinence is often the prominent symptom of cystocele).

The transposition operation is ideal for the accomplishment of these two objects and the results obtained have shown it to be ideal in a

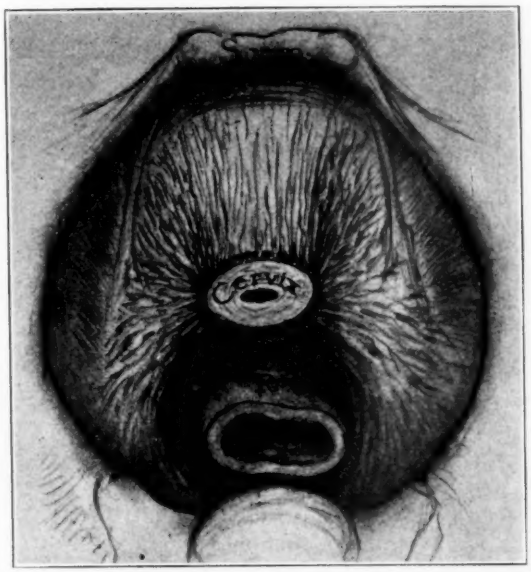

Fig. 2.—Vesico-vaginal fascia viewed from above. Dotted line common site of tear (from Frank).

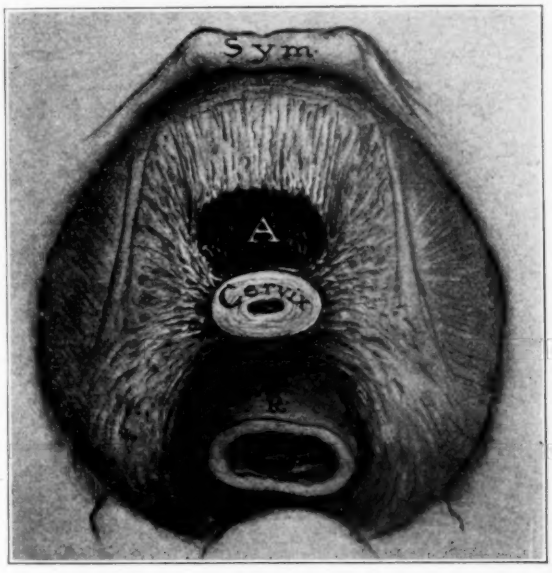

Fig. 3.—Hernial ring of cystocele shown from above.

limited class of cases. The limitations and modifications of the transposition operation have been discussed in former papers.

A firm closure can be obtained in a large majority of cases by the advancement operation with plastic repair of the broad ligaments. Goffe did pioneer work in establishing the advancement operation. The closure as illustrated (Fig. 4) should restore the torn edges of the fascia to the cervix. The bases of the broad ligaments on either

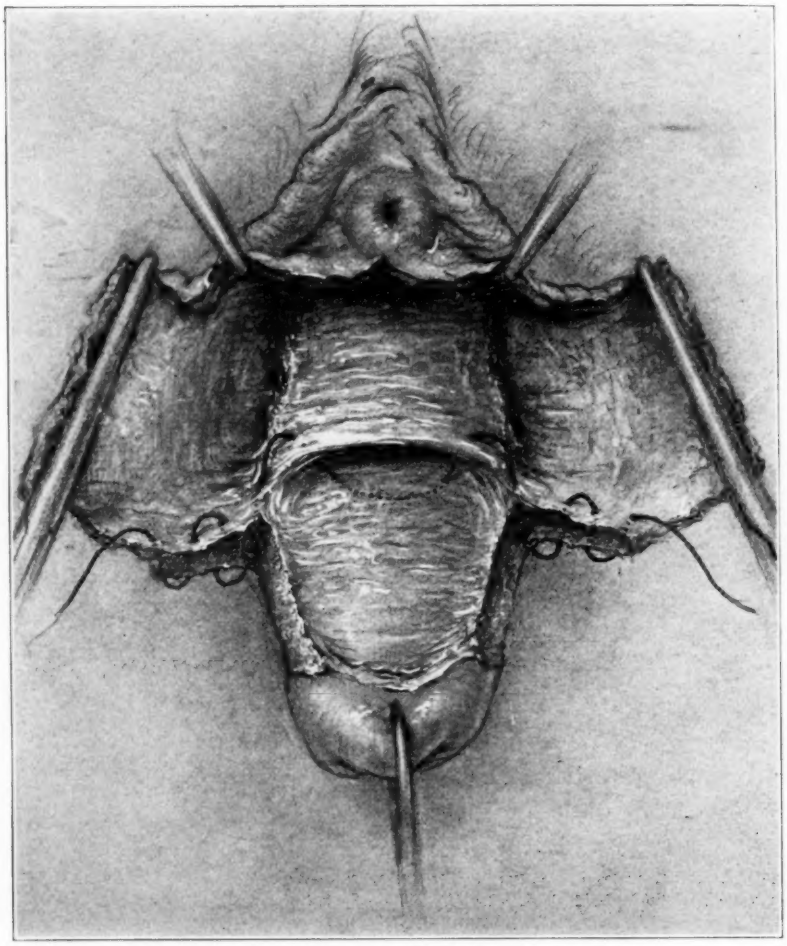

Fig. 4.—Suture of the free edges of the vaginal flaps to the cervix at about level of internal os, closing hernial opening.

side have been clamped and incised and fascial flaps have been reflected from the vaginal wall on either side. The hernial opening is closed by suture of the free edges of the vaginal flap to the cervix at about the level of the internal os. One or two sutures for this purpose are generally inserted before the broad ligament clamps are removed but are not tied, as tying them may interfere with accurate suture of the free edges of the broad ligament. The suture illus-

trated is inserted for the purpose of controlling the bleeding from the broad ligament and for the purpose of approximating the free edges of the broad ligament in front of the cervix. The sutures for closure of the hernia are first tied and then the sutures which include the cut ends of the broad ligaments are tied. One or two additional chromic catgut sutures are inserted if need be to obtain firm closure

Fig. 5.—Rectocele and cystocele.

of the hernia. It will be found that by this means the urethral canal has been restored to its normal position and the cure of urinary incontinence is assured. Care should be used that excessive traction is not made upon the urethra. This can be obviated by seeing that the urethra is left only in a relative state of fixation. (The amount of mobility of the urethra is generally proportionate to the extent of the urethrocele.)

Some plastic work is frequently required upon the cervix. This may consist in excision of the anterior lip or in amputation of the cervix, depending upon the requirements in the individual case. The operation is completed by the excision of any redundant mucosa and closure of the mucous membrane wound. In making the mucous membrane closure it is well to include some of the fascia or cervix in each suture for the purpose of obtaining increased firmness.

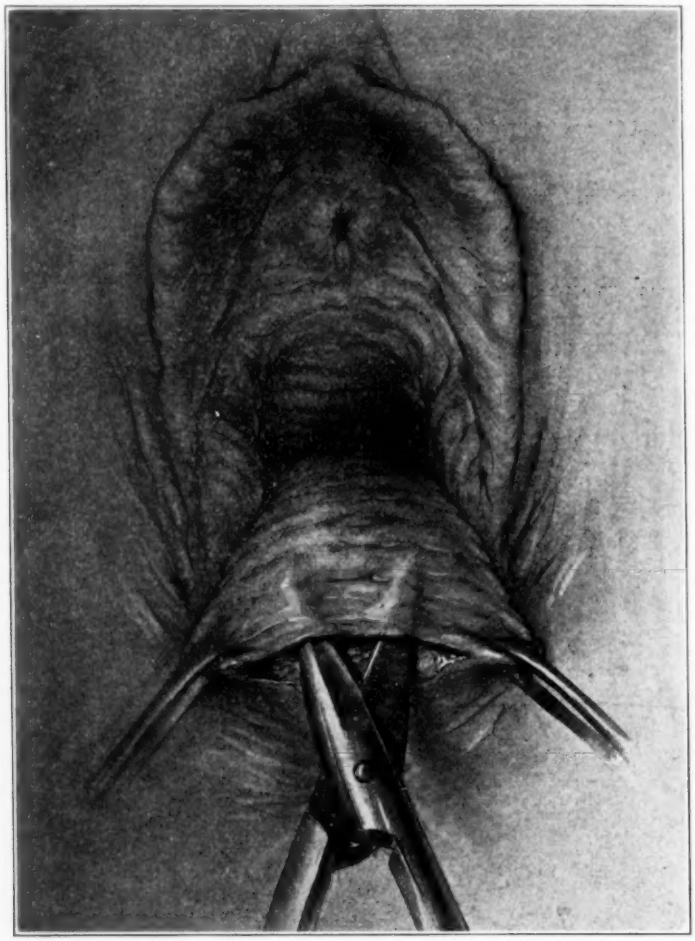

Fig. 6.—Blunt dissection of vaginal wall over rectocele.

In complete prolapse the two objects mentioned above can be accomplished by the Mayo operation or by a modified transposition operation. Personally we prefer to make in such cases a rather extensive plastic on the broad ligaments and to excise much of the uterus if large, leaving only enough of the lower posterior uterine wall to form a floor for the bladder. This permits leaving consider-

able of the broad ligaments intact and the result is not jeopardized in case of infection.

HIGH RECTOCELE

Experience with thorough dissection in cases of high rectocele has impressed me with the fact that the nature of the injury is like the

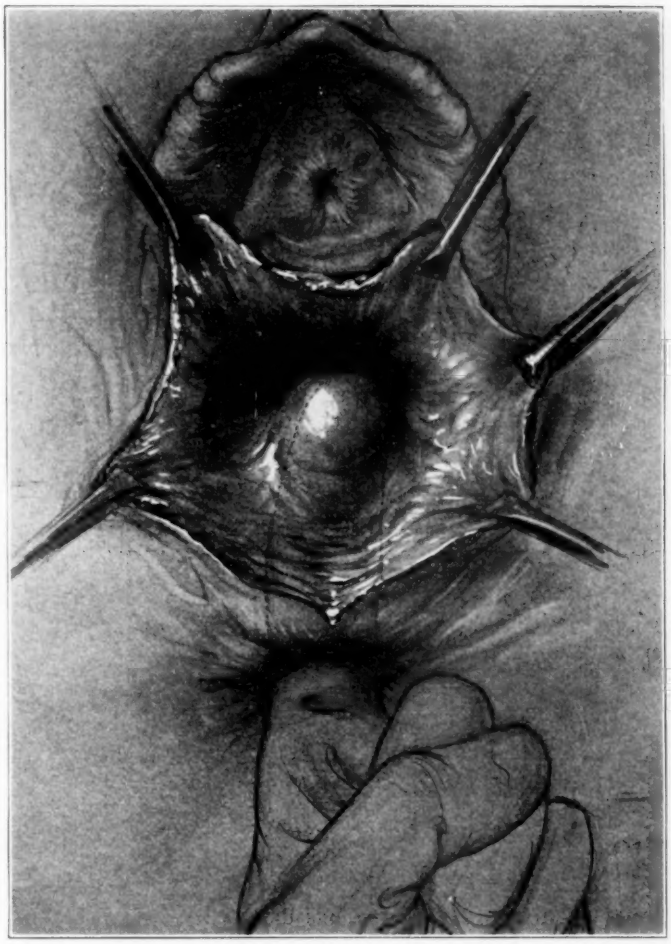

Fig. 7.—Hernial opening in rectocele.

one found in cystocele. By means of this dissection we have been able to outline the nature of the fascial rent and have found that the fascia is torn from the cervix and broad ligaments much as it is in cases of cystocele. During labor the tension upon this fascia is the result of displacement of the perineum below the pubes and the tension is consequently longitudinal and the result is a transverse tear. If the rectocele is thoroughly dissected it is easy to estimate by

approximation of the freed edges the site of the lesion. The edges of the hernial ring can be carried up to the cervix and fixed without undue tension. When this is done the result is that the perineal body is pulled forward toward the pubes in the line of its natural location. Fig. 5 is a high rectocele from which these illustrations were made.* Fig. 6 illustrates the technic of blunt dissection. This

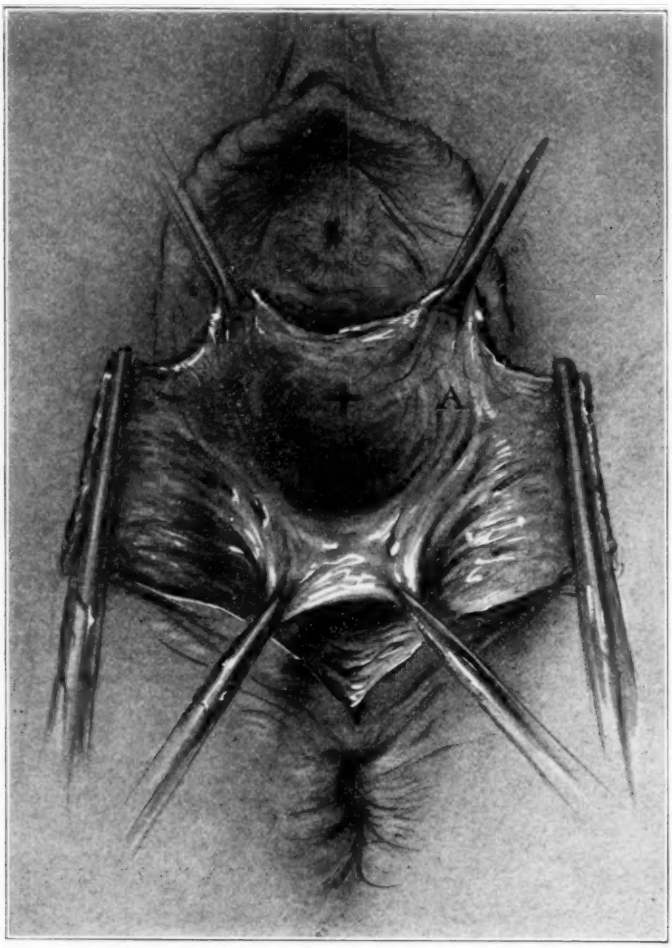

Fig. 8.— + Indicates location of cervix. *A*, Broad ligament on either side. Torn edges of fascia retracted by forceps. Rectal hernia.

is made by making consecutive short median longitudinal incisions and by keeping the scissors as near to the mucous surface as possible, a procedure which minimizes the amount of injury to the recto-vaginal fascia. In Fig. 7 the dissection has been carried up to the cervix. The vaginal flaps are held apart and the finger in the rectum

*The drawings were made from life and accurately illustrate the lesion.

demonstrates the nature of the hernial opening. In Fig. 8 all the fascia has been carefully dissected from the mucous flaps. The freed edges are put on tension so as to illustrate the amount of rent in the fascia. The free edges of the fascia can be carried up to the cervix without undue tension, which means that this fascia has been torn from the base of the broad ligaments and from the posterior wall to

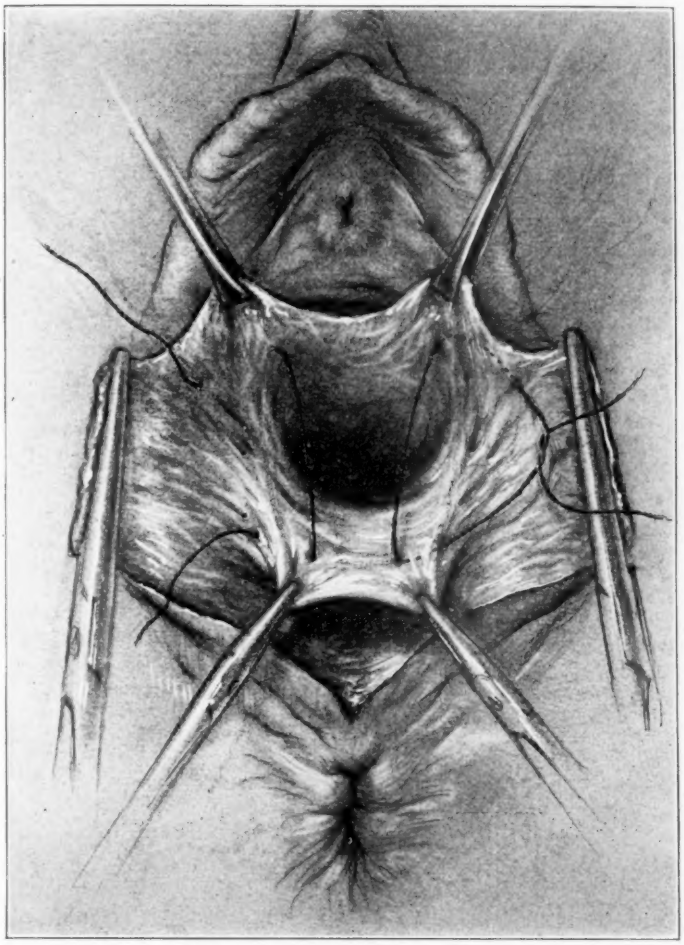

Fig. 9.—Torn edges of fascia flaps are sutured to the broad ligament and posterior to the cervix.

the cervix. Fig. 9 shows the suture which restores the free edges of the hernial ring to the base of the broad ligaments. In bad cases we frequently use a linen suture and in moderate cases chromic catgut sutures. Additional sutures are inserted as needed for firm closure of the hernia. These sutures should be so inserted that when tied they are not on undue tension. Fig. 10 shows the hernia closed. The

operation is completed by excision of any redundant mucosa and by subcutaneous suture with plain catgut. When the operation is completed rectal examination will show a fairly firm wall, much like the normal, extending from the anus to the cervix.

High rectocele has received considerable attention of late and some excellent papers have been presented by Ward, Spaulding and Frank.

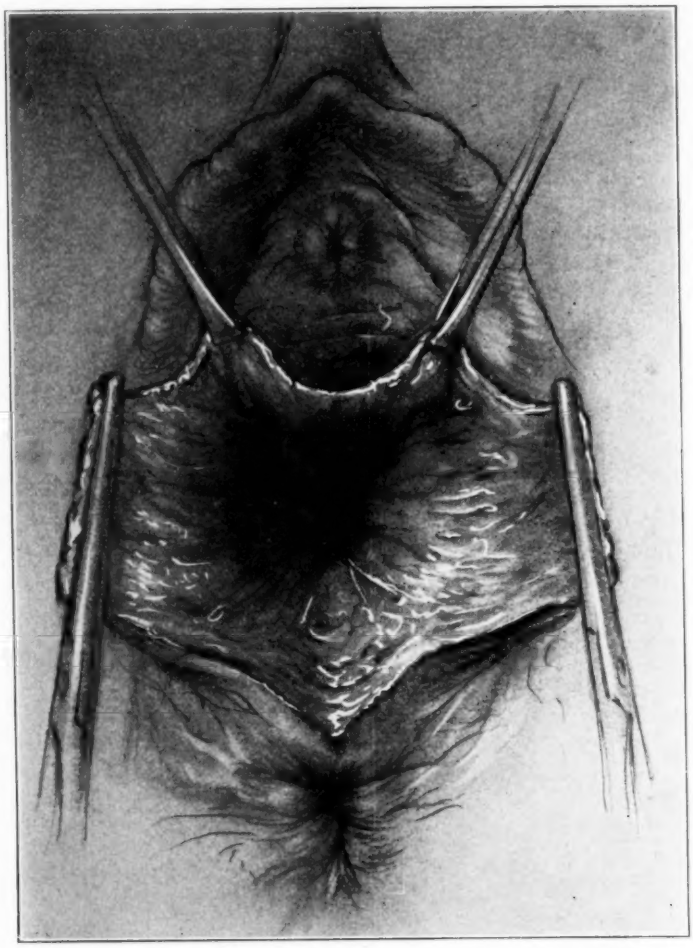

Fig. 10.—Hernial opening closed.

The obliteration of the deep peritoneal pouch according to the method of Ward will probably prove to be of great value in selective cases. My experience with this operation dates from 1919.[1] The results have been very encouraging and have been improved much of late since a more thorough dissection has been made.

[1]Surgical Clinics of Chicago, October 1919, page 1231.

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SUMMARY

Cystocele is hernia resulting from a transverse tear in the vesicovaginal fascia. The tear is almost universally from near the site of the cervix.

Closure of the ring and restoration of the fascia to its normal location will cure the urethrocele.

Plastic repair of the broad ligaments insures firm closure of the ring.

The operation is simple if treated as a distinct hernia and failures should generally mean faulty technic or judgment.

High rectocele is a transverse tear in the rectovaginal fascia. The site of the lesion is near the cervix. With thorough dissection the torn edges of the fascia can be easily exposed and sutured into normal position.

In perineal lacerations the fascia and muscles are also often injured near the rami of the pubes but this class of lesion is not presented for discussion at this time.

104 South Michigan Avenue. (*For discussion, see p.* 487.)

RELATION BETWEEN PLACENTA AND THE SECRETION OF MILK*

By Professor Dr. Oskar Frankl, University of Vienna, Austria

IN SPITE of many investigations and studies the question of the relation of the placenta to lactation has remained an unsolved problem. We know that the placenta during pregnancy, presumably in conjunction with ovarian function, is responsible for the hyperemia and development of the mammary gland and the formation of colostrum. True milk, however, appears only after the expulsion of the fetus. It remained an open question whether in this connection the placenta though acting as a stimulating factor on the breast gland simultaneously inhibits the secretion of milk. Obviously this question is closely connected with the mooted problem whether the placenta can be regarded an organ endowed with an endocrine function.

Adler, Thaler and Frommer demonstrated that under the combined effect of pregnancy and the injection of placental extracts animals are liable to manifest a subtetanic state. It is a well-known fact that an intervening pregnancy checks the function of lactating breast glands. Such facts make it very probable that the placenta is producing some substance with a definite biochemic effect on the organism. But such experiments are not entirely convincing and it became necessary to resort to different types of investigation.

*Read at the Forty-eighth Annual Meeting of the American Gynecological Society, Hot Springs, Va., May 21-23, 1923.

That the fetus is not the deciding factor in the secretion of milk is proved by active lactation subsequent to the expulsion of a hydatiform mole or a dead fetus. Ovarian function in relation to lactation is distinguished by the fact, on the one hand, that castration performed during pregnancy does not preclude lactation, while, on the other hand, in the experience of animal breeders castration is successfully resorted to for the purpose of prolonging for a longer time lactation in the mother animal.

The influence of the nervous system on lactation cannot be regarded as deciding because, in the human, normally functionating breasts have been observed in cases of complete transverse lesions of the cord and, in animals, normal milk secretion in mammae successfully transplanted into other regions. In this respect convincing, prove also experiments on artificially united (parabiotic) animals and the observation made on the pyopagic sisters Blazek, carefully studied in our clinic in Vienna. In experiments on animals and this one observation on the human typical milk secretion was seen after delivery of the pregnant twin, also in the other nonpregnant conjoined individual.

Many attempts are recorded in literature of producing secretion of true milk by the injection of placental extracts and of emulsions of placental tissue, with varying results. It is noteworthy that similar results were obtained by other investigators with the injection of extracts prepared from tissues other than placenta. Cristea and Aschner were successful in reestablishing a previously existing milk secretion by means of various lymphagogues. The objections made frequently that all such experiments with the injections of protein bodies cannot well be compared with the physiologic function of an organically connected organ like the placenta, certainly are fully justified.

Halban as the first claimed that it is directly the loss of the placenta which changes the secretion of colostrum into one of milk. To prove or disprove this theory I designed certain experiments, consisting in the transplantation of the placenta of a mouse under the skin on the back of another mouse. Such grafts in general led to three possible end-results. In case of an intervening infection a large abscess formed destroying the implanted placenta. In other cases, aseptically accomplished, the graft did not take and the placenta became rapidly necrotic. In the third, successful group, the implanted placenta established satisfactory connection with the adjoining tissues and the organ could be observed to persist for about three to four weeks. After this time histological studies show a complete absorption of the grafted placenta.

In these experiments the placentas of pregnant mice were employed within ten or twelve days of full term and transplanted under the

skin of the back of mice approximately in the same stage of pregnancy. In this third group of successful transplants the following results were observed: Mice, from a strain known to be able to nurse satisfactorily their young, would have litters of five or six. The newborns would seem normal in their appearance and would immediately begin to suck the mother animal. But they invariably died within the next five to seven days, evidently from starvation. Investigations showed that the mamma of the mother animal was secreting colostrum and not milk. In the unsuccessful transplants of the first and second groups lactation proceeded normally. *A successful transplantation of a placenta on a pregnant animal causes persistence of colostrum secretion.*

These same animals were permitted to become pregnant again and again they were able to nurse their young.

These experiments, therefore prove clearly that in animals, known to have ample milk, the effective grafting of placenta temporarily hinders milk secretion, evidently under the influence of an active hormone supplied by the living placenta.

This ability of the placenta to prepare during pregnancy the mamma for its secretory function but at the same time to inhibit actual function has its perfect analogy in the function of the corpus luteum. Also the yellow body through its hormones prepares the endometrium for menstruation by causing the typical premenstrual swelling, but only the involution of the corpus luteum, which follows the death of the ovum, determines the appearance of the menstrual flow.

Of special interest proved to me a recent article in the AMERICAN JOURNAL OF OBSTETRICS AND GYNECOLOGY by Stimson. He showed that the retained portions of the placenta could hinder milk secretion, which developed normally immediately after the removal of the retained tissue. This observation corresponds exactly to the results of my own experiments on animals. Also this clinical experience points clearly to the fact that the placenta must be regarded an organ endowed with endocrine activity, whose function it is during pregnancy to prepare the breast glands for the secretion of milk and simultaneously to prevent secretory activity.

ERSTE FRAUENKLINIK, VIENNA, AUSTRIA. (*For discussion, see p.* 489.)

FOLLOW-UP RESULTS OF 908 CASES OF UTERINE CANCER TREATED BY RADIUM*

By Harold Bailey, M.D., and William P. Healy, M.D.,
New York City. N. Y.

WE have used radium in the treatment of uterine cancer at the Memorial Hospital for over eight years and from time to time we have reported our results. In May, 1921, at the last presentation, it was apparent that there was considerable improvement in the results of the second three-year period and we ascribed this improvement in the years 1918 to 1920 to the difference in our methods of treatment. To some extent this still holds good. In the first three years, 1915, 1916, and 1917, the treatment was conducted with a limited amount of radium and its application was localized as in the Continental procedure. At the end of the year 1915, our attempts to cross-fire through the rectum and through the uterus were discontinued because of the frequency of rectovaginal fistulae.

The technic adopted in 1916, which we still think well of from the standpoint of those operators using amounts comparable to 100 mg., consisted in the reapplication of the radium to different areas in the cervix. The first week 1200 millicurie hours were used, the following week the same dose and the third and fourth weeks a slightly smaller dose. This always leads to a deep slough in the cervix, but it concentrates the radium in the center of the disease.

In some thirty cases use was made of the Percy operation with the radium application two weeks after the burning, but it was finally given up because the majority of the cases developed fistulae. Of the thirty Percy cases, one patient treated in 1915 is well today. Another patient, burned by Dr. Le Roy Broun and sent to us in 1915 for radium treatment, is well; and a third who had two cauterizations and was later treated by us in 1916, was well at the time of writing this report. However, we believe that the interference with the blood supply in addition to the cauterization and the tendency of the operator to burn too extensively, together with the thinness of the barrier for filtering the radium that is afterwards applied, renders the operation inapplicable in most cases.

From the year 1918 to date we have made great use of cross-firing from the vagina, from the neck of the uterus and from the external surface of the body and practically all our cases have been so treated during this last period of five years.[1] The particular method devel-

*Read at the Forty-eighth Annual Meeting of the American Gynecological Society, Hot Springs, Va., May 21-23, 1923.

oped was the use of a platinum piece in the cervix with another above in the neck of the uterus for a total of about 3000 millicurie hours and the radiation of the parametrium by a mass of radium equal to a gram or more placed in the vault of the vagina in the so-called bomb container which is directed toward each lateral fornix in turn, and a central application against the cervix to reinforce the amount of radiation in the tissues immediately adjoining the cervix. The external application was given by radium on a block applicator placed in six different regions around the pelvic girdle, a half skin dose being given in each place. We found that when a full erythma dose was given with additional irradiation from below, the skin area would be severely burned. Lately, that is in the past year, we have substituted for the block which gives only a small dose deep in the pelvis, x-ray with prolonged and intensive application. We believe that some form of external irradiation is necessary and advisable in most cases.

Our technic with x-ray has been to give the irradiation through four areas, two on the front and two on the back of the body and so arranged that the entire skin surface over the pelvis is covered. Two tubes working simultaneously and so placed that the rays converge toward the center of the pelvis result in considerable cross-firing beneath the skin surface. The first application is for 15 minutes, the second occurs 48 hours later for the same length of time. The filter is 5 millimeters of aluminum, the focal distance 12 inches, the spark-gap 10 inches and the kilovolts 90 to 110 with 5 milliamperes of current. The additional irradiation thus provided is of considerable value in the treatment of the areas involved by cancer that are some distance from the cervix and vaginal vault.

The follow-up has been particularly difficult because in the earlier years we were working in a new field and the length of time of the action of the radium, the depth to which efficient irradiation is carried and the later results from the production of fibroid tissue were all unknown to us, and these patients had to be brought in very frequently for observation. As the years passed these women were asked to return to conferences for the instruction and information of visitors from this country and from abroad. After four or five years of this frequent following and control, the women objected decidedly and in a few instances it is now impossible to get them to come back. Some even seek to avoid observation and recall by refusing to answer letters and moving to new addresses without informing the hospital. A very personal request is necessary for those who have been more than five years in the follow-up service; a nursing call or letter is rarely effective. However, without the Social Service Workers, we would be in a very poor position to continue our work; their energy and tact have helped to make this report possible. Most of the

cases of the first four years have been seen by us, but from a number we have accepted, as evidence of their present condition, either a personal letter or a letter from their physician, stating that their health was satisfactory. Those cases that have been lost, and there are quite a number of them, have been accepted as a charge against us, in other words they have been considered as dead. Occasionally after one of these reports a case which has been placed in the final list returns, but this is unusual and does not interfere to any extent with the statistics.

Advanced Primary Cervical Cancer.—In this group there was very little hope of producing a cure under any circumstances. Not only was the cervix involved but the parametrium as well when the treatment was undertaken. In the first two or three years all the advanced cases were treated whereas now we limit ourselves to those cases in which there is apparently an opportunity to irradiate beyond the outlying part of the tumor growth. We are averse to treating the very advanced cancer with radium even with the so-called palliative treatment. It seals up the surface of the growth and there is no outlet for the broken-down areas beyond. To give a heavy treatment such as we use in the other cases to one of these women produces a condition of pain and distress that is beyond description.

The advanced primary cancer forms the largest group of our patients. If we take the first three years, 1915, 1916, and 1917 in which the radiation was without any decided amount of cross-firing there were in all 80 cases and but two women are alive. In 1918 with the use of the bomb and block there were 41 cases, and six women, or 14.5 per cent, are alive and free from the disease today. In 1919 there were 69 cases with but 5 or 7 per cent well and two alive with the disease progressing. In 1920 there were 92 cases with 8 or 8.7 per cent alive and no evidence of cancer, and seven alive with symptoms.

In 1921, there were 85 cases, and 12, or 14 per cent, have no evidence, and 19 are in various stages of the disease. In 1922 there were 80 cases, and 12, or 15 per cent, are apparently well, and 45 others are alive. While not enough time has elapsed since the treatment of the cases of the last two years to give any idea of the final results, still of the 165 cases that were beyond the aid of surgery, 24 cases now show no clinical signs of cancer.

Borderline Cervical Cancer really laps over into the advanced group and the line of demarcation is a clinical one and largely influenced by the personal equation of the examiner. In general, in our opinion, these cases were not suitable for surgery. *In the first five years* there were 33 cases of which 8, or 24 per cent, are alive. All but one of these are known to be free of evidence of the disease. In the last three years there were 51 cases and of these there are 22, or

43 per cent, with no evidence and 15 show the presence of tumor tissue.

In the *early operable group* there were 11 cases treated previous to January 1st, 1919. Of these, 3, or 27 per cent, are alive and free from evidence of cancer for five years or more. If the rate is corrected by deducting 3 deaths from intercurrent disease and one following operation in another clinic the result is 43 per cent clinically cured for the period. In the last four years there were 48 cases and of these 32, or 66 per cent, are free from evidence of the disease. If we are permitted to deduct 3 cases, one dying after an operation in another hospital a week after treatment and two others giving false addresses so that they could not be followed, there are 32 alive of 45 cases or 71 per cent. In these early operable cases we naturally look for and hope to get excellent results from irradiation and very few of our cases receive any other treatment.

However, there seems to be no doubt that hysterectomy alone has cured cases of cancer of the cervix and for that reason it may, at times, be considered advisable, in very favorable cases, to follow the full irradiation treatment by an abdominal hysterectomy some weeks later so that the patient will not be deprived of any advantage which may accrue from such procedure. Just how much is to be gained by this is still a matter of conjecture.

Recurrent Cancer.—Previous to 1918, there were 52 cases and 2 are alive and well and one other is alive but with some evidence of tumor. In the past five years during which time the technic has been considerably elaborated by the use of cross-firing and the embedding of radium emanation in the lesion, a remarkable number of these cases has been apparently cured. There were 168 cases in this group and 38, or 22 per cent, have no clinical evidence of cancer.

Prophylaxis Following Hysterectomy.—This is a very interesting group made up entirely of cases referred by various operators soon after abdominal panhysterectomy had been done in each case. In no instance was there any evidence of cancer when the patient presented herself to us for treatment; therefore it seemed probable that the best procedure would be to irradiate the entire pelvis by means of cross-firing with the bomb placed within the vagina and the radium block or more recently the x-ray for deep external radiation. There were 29 cases from 1917 to 1923 and of these 21, or 72.5 per cent, are alive and well and have remained free from any sign of recurrence and five of these cases have been well for five years.

Cancer of the Body of the Uterus.—We have had 41 cases of this type of which 21, or 49 per cent, are alive and well. Three of these have passed the five-year period. Nine of the 21 cases had hysterectomy following the irradiation. In the other cases hysterectomy was

not considered advisable because of other complications or constitutional diseases and these cases have been treated by irradiation alone. The plan followed was the introduction of radium into the uterine cavity for 3000 to 4000 millicurie hours with light filtration and external radiation with x-rays. Despite this treatment in nearly every instance subjected only to irradiation evidence of the disease reappeared within a year.

FOLLOW UP OF 908 CASES OF UTERINE CANCER TREATED BY RADIUM TO JANUARY 1ST, 1923*

	ADVANCED PRIMARY CERVIX	BORDERLINE CERVIX	EARLY OPERABLE CERVIX	RECURRENT	BODY OF UTERUS	PROPHYLAXIS AFTER HYSTERECTOMY	PERCY
1915	15	0	1 (1)	18 (1)	1 (1)	0	15 (2)
1916	24 (1)	3 (0)	3 (0)	9 (1)	1 (0)	0	11 (1)
1917	41 (1)	3 (1)	3 (1)	26 (1)	0	2 (0)	
1918	41 (6)	17 (5)	4 (1)	35 (4)	7 (2)	8 (6)	
1919	69 (11)	10 (2)	9 (6)	43 (5)	5 (1)	4 (3)	
1920	92 (14)	12 (3)	13 (4)	37 (14)	5 (3)	10 (8)	
1921	85 (31)	12 (9)	10 (8)	24 (14)	9 (8)	3 (3)	
1922	80 (57)	27 (26)	16 (16)	29 (25)	13 (12)	2 (2)	

*Figures in the parentheses represent the number of cases alive January 1, 1923.

These results, we believe are sufficiently encouraging to be worthy of careful consideration. It is our opinion that they could not have been obtained without the use of massive doses of radium and without thoroughly radiating the parametrium.

REFERENCE

(1) *Bailey and Quimby,* AM. JOUR. OBST. AND GYNEC., February, 1922, iii, No. 2.

MEMORIAL HOSPITAL.

(*For discussion, see p.* 491.)

CONTRIBUTIONS TO THE PATHOLOGY OF VULVAR DISEASES

A, Angiomatous Tumors of the Vulva; B, Abscess of the Clitoris; C, Granuloma Inguinale; D, Leucoplakic Vulvitis

By Fred J. Taussig, M.D., St. Louis, Mo.

DURING the past few years it has been my good fortune to see a number of interesting conditions about the vulva concerning which but little has been written. A brief clinical history of these cases together with the attendant pathology has seemed worth while placing on record. I have also during this time had occasion to see and study the histopathology of an additional series of cases of leucoplakic vulvitis, some of which were associated with vulval cancer and have summarized my present views upon this disease.

A. *Angiomatous Tumors of the Vulva.*—It would seem that most of these tumors develop on the basis of a congenital nevus. Hemangiomata are most frequently found in little children (as seen in the reports of Kennig, Guyot and Saenger). Occasionally they are associated with telangiectatic swellings. Lymphangioma of the vulva also has been described by Brindeau, Epstein and Duret but the term lymphangioma should be limited to the definitely circumscribed tumors. An illustration of these types can be seen in the following:

1. *Hemangioma* in a baby four months old. At birth a flat nevus was observed in the region of the left labium majus. It grew steadily and at the time of operation was 15 × 15 mm. in size and 2 mm. above the level of the skin. The skin showed a slight tendency to ulceration. The growth was excised and microscopic examination showed a typical hemangioma.

2. *Telangiectatic Angioma of the Clitoris and Labium.*—This patient was twenty-eight years old, unmarried, and according to the statement of her aunt, was born with a birthmark at the site of the vulval tumor. Since puberty it had grown larger and repeated slight bleeding had occurred. Not until three weeks previous to her admission to Barnes Hospital had there been any pain. The entire right labia at this time were converted into a mass the size of a hen's egg consisting of a network of varicose veins. The clitoris formed a blue swelling the size of an acorn. The left side was unaffected. The tumor removed at operation showed on microscopic section numerous telangiectatic veins (Figs. 1 and 2).

*Read at the Forty-eighth Annual Meeting of the American Gynecological Society, Hot Springs, Va., May 21-23, 1923.

3. *Lymphangiectatic Hypertrophy of the Vulva on a Basis of Nevus Unius Lateralis.*—This unique condition seen in a girl sixteen years old at the Barnard Free Skin and Cancer Hospital was also congenital. A small reddish area was noted by her mother at birth. Eighteen months later it became more prominent. The tumor grew more rapidly after the age of thirteen when menstruation began and continued at intervals of from 6 to 8 weeks. Salves and washes had been employed by the girl to relieve the irritation produced by the profuse milky-white vulval discharge.

On examination the right labium majus and minus together with

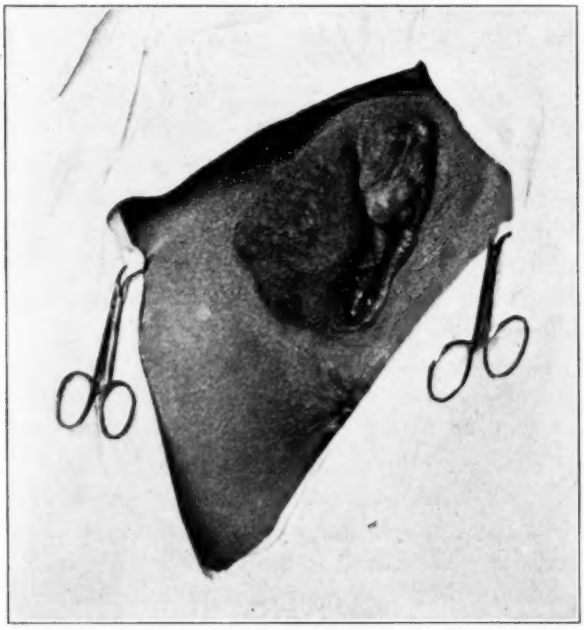

Fig. 1.—Telangiectatic angioma of the right labia and clitoris.

half of the clitoris was converted into a thickened irregular swelling with a few warty projections about the prepuce (Fig. 3). The peri-anal skin on the right side was also thickened and showed papillary excrescenses. Most striking was the exactness with which the lesion was limited to the right side. The lesion in the crural folds was, as confirmed by biopsy, only a secondary dermatitis resulting from the irritating lymphorrheal discharge. Of further interest was the fact that a unilateral nevus was also present upon the lips and tongue of this girl. The hypertrophic labial mass was removed surgically and microscopic section showed a combination of condylomatous epithelial proliferations with marked lymphangiectatic swell-

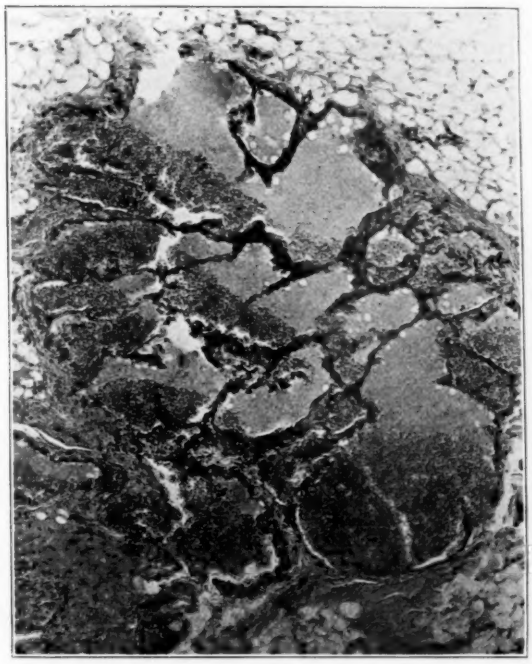

Fig. 2.—Telangiectatic angioma (microphotograph).

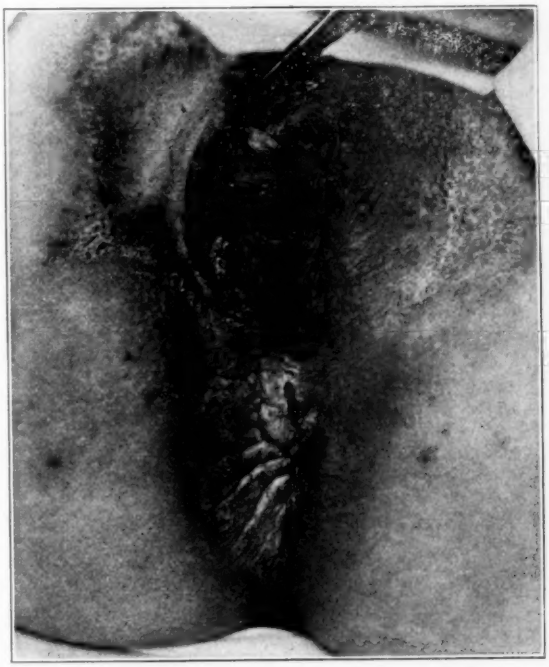

Fig. 3.—Lymphangiectatic hypertrophy of the labia on a basis of nevus unius lateralis.

Fig. 4*A*.—Lymphangiectatic hypertrophy of the vulva. Area outlined in broken lines is photographed with higher magnification in Fig. 4*B*.

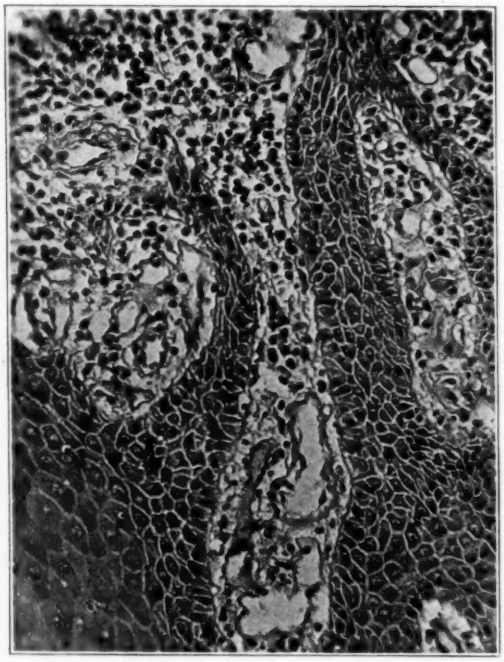

Fig. 4*B*.—High power photograph of the area outlined in Fig. 4*A*.

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ing in the subjacent dermis (Figs. 4*A* and *B*). A more detailed description of this case will be published later.

B. *Abscess of the Clitoris.*—While abscesses of considerable size not infrequently develop from furunculosis and bartholinitis, I have found no record of any extended suppurative process involving primarily any portion of the clitoris. The following case is of interest not merely because an abscess was situated in this unusual site, but also because it seemed definitely responsible as the chief focus of infection for a chronic arthritis from which the patient has been suffering many years.

Mrs. A. B., forty-six years old, 2-para, had had a small abscess in the upper left vulval region lanced eight years previously. There was no history of gonorrhea and the scar from this incision was found on a line with the clitoris and 2 cm. to its left. The patient stated that while she experienced no further pain in this region after the incision, there was at times occasional discomfort with a slight offensive discharge the source of which she could not determine. During these eight years symptoms of chronic arthritis developed with occasional slight elevations of temperature. A search for the primary focus had been directed everywhere except the genital region. Finally on August 4, 1922, after a lapse of eight years, a fever of 103° with vague abdominal pains suggested a possible appendicitis. The fever remained high for 5 days, then the abdominal pains subsided and suddenly acute pain in the vulval region was experienced. I was called in consultation and found a small hard mass the size of an acorn in the region of the left crus of the clitoris directly beneath the scar of the former incision. Hot poultices within 36 hours led to the formation of an abscess the size of a walnut at this point.

A few drops of pus escaped from a pinpoint opening near the old scar. The main abscess sac, however, was still deeply situated. and, in view of the history of prolonged infection, it was deemed best to remove the entire mass, instead of merely incising the abscess. The accompanying photograph (Fig. 5) shows clearly the site of this abscess. The cross-section (Fig. 6) indicates the thickness of the abscess sac, but since about one ounce of pus escaped from it before the tissues were hardened, the cavity is much smaller than at the time of removal. Microscopic sections of the lining (Fig. 7) showed a pyogenic membrane without any visible epithelial elements and a diffuse suppurative process infiltrating the connective tissue wall.

C. *Granuloma Inguinale.*—A more careful analysis of certain types of chronic ulcerations about the genitals in the negro race, that have in the past been termed chronic chancroids, lupus or rodent ulcer, show that a certain group of these cases are characterized by extensive superficial ulcers extending up into the inguinal region, a fairly typical reaction to intravenous administration of tartar emetic, and

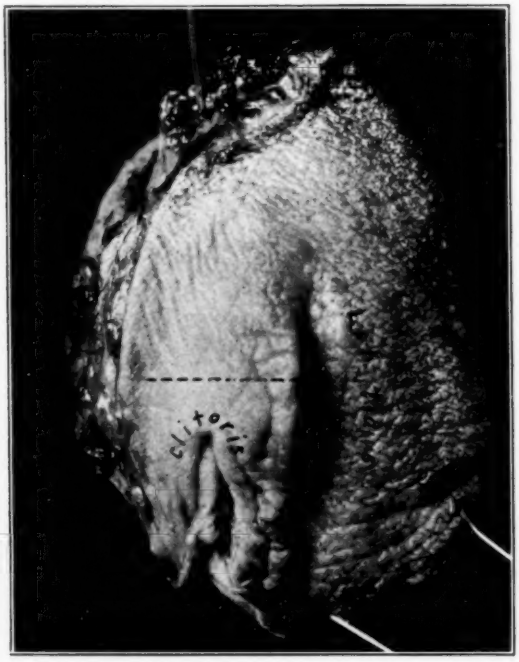

Fig. 5.—Abscess of the clitoris, specimen removed at operation.

Fig. 6.—Cross section of abscess at a point shown in dotted line on Fig. 5. The abscess cavity is collapsed. It contained about 30 to 50 c.c. of a thick pus.

the presence of bacteria termed Donovan's bodies in the secretions and tissues of the ulcer. While further investigations are still required for positive proof of the etiology of this disease, I think we can accept "granuloma inguinale" as a definite clinical entity. The recent reports of Randall, Small and Belk are particularly convincing.

I am able to contribute a brief report of two cases to the literature on this subject.

1. The first case was observed by Dr. E. F. Schmitz to whom I am indebted for the permission to include it in this report. This patient, a negress, was so dull mentally that no history could be obtained from her further than the fact that the sores have been present for several years and that antiluetic treatment, cauterization and local

Fig. 7.—Abscess of the clitoris. Pyogenic membrane lining the cavity.

applications had failed to prevent their gradual extension. She was a negress of about thirty years of age. She had no other physical ailments. The ulcers were typical of "granuloma inguinale." Injections of tartar emetic were given with marked improvement. The edges showed definite healing. Unfortunately, ten days after her admission to the City Hospital, following the fourth injection of tartar emetic the patient suddenly died. An autopsy could not be obtained.

2. In the second case the record is more complete. This patient, a negress twenty-eight years old, had had two children, both delivered by cesarean section, one of which was done by me in 1916. At this time the vulva was normal. Menses were regular up to three years ago when an ulcerative process began about the vulva. Wassermann reaction was repeatedly negative. Ducrey bacilli were ab-

sent. The ulceration gradually extended from the vulva and perineum to the left inguinal fold, its edges irregularly outlined, not undermined, the ulcer very shallow and its base for the most part smooth and clean. For the past few months a similar superficial ulcer had appeared upon the lower lip of the mouth. A biopsy specimen was obtained from the lip and vulvar ulcers and showed identical lesions, merely a chronic granuloma without giant cells, tubercles or evidence of epithelial proliferations. In smears from the secretion and in some of the sections typical Donovan bodies were found. Injections of tartar emetic given intravenously caused improvement but, as yet, two months after treatment was begun, the ulcers have not entirely healed. A full report of the case will be given elsewhere.

D. *Leucoplakic Vulvitis.*—In a previous article* I analyzed 21 cases of leucoplakic vulvitis seen up to 1919. In the past four years an additional 19 cases have come under my observation, making a total of 40 in all. A number of these patients were referred to me by my colleagues, Drs. Ehrenfest, Gellhorn, Engman, Mook and O. Schwarz, otherwise it would have been impossible for me to have collected such a considerable material for study in these ten years. I think I am right in saying that this is the largest material that has as yet been utilized for a clinical and pathologic investigation of this disease. While I have not fully completed the histologic portion of this work, I can at the present time give a brief summary of my conclusions. Material for the histologic study was obtained in 29 out of the 40 cases, in 25 by complete vulvectomy, in the remaining four by biopsy or autopsy excisions. In 18 cases (45 per cent) the leucoplakic vulvitis was complicated by a carcinoma. If in my first series the incidence of carcinoma was 66 per cent, while in the series since 1919, the incidence of carcinoma was only 21 per cent, this may in part be explained by the fact that the profession and to some extent also the laity have learned to recognize leucoplakic vulvitis as a so-called precancerous condition and more radical measures have been used to cure this disease before a cancer developed. In fact, I am convinced that if the laity and profession were cognizant of this tendency of leucoplakic vulvitis to stimulate a cancer and excised the affected area, the incidence of vulval cancer could be cut down almost one half. During the past 11 years I have seen 36 cases of cancer of the vulva, 18 of which showed evidence of having developed on a basis of leucoplakic vulvitis.

Nicknames cling to diseases as they do to human beings. I suppose it will be many years before the term kraurosis vulvae will be dropped from our textbooks. Unfortunately Breisky in his first twelve cases saw such a high proportion in which the vaginal outlet

*Arch. Dermat. and Syph., June, 1920, n. s., I, 621-635.

was constricted, that he considered this constriction as the main feature of the disease and hence employed the term kraurosis (to shrivel up) to distinguish it. He did not appreciate that the same clinical pathologic picture could be present without such constriction and that the constriction could occur at times without any evidence of the characteristic histologic changes he had found.

Kraurosis, therefore, is a symptom and not a disease. If we use the term at all, it should be limited to that simple form of atrophic vulvitis resembling in its pathology and course atrophic vaginitis which is unattended by leucoplakic changes. I would prefer to call this condition atrophy sclerosis of the vulva, and so eliminate the ambiguity that still clings to the term kraurosis.

In this series of 40 cases of leucoplakic vulvitis, the symptom of kraurosis was present 16 times and always only in these women in whom the entire vulva was involved in the leucoplakic change. It is evident that the formation of a kraurosis must depend partly on the condition of the vaginal outlet previous to the onset of the disease. In a multipara with relaxed outlet the most extensive leucoplakic vulvitis will not produce kraurosis. In nulliparae or virgins, on the other hand, such a constriction is readily formed.

We may distinguish two types of leucoplakic vulvitis, one in which the affected area is one-sided and somewhat irregular in shape, the other in which the entire vulval ring is symmetrically involved. In my series of 40, nine were localized and the remaining 31 were of the symmetrical or complete type. Of these 31 symmetrical cases, the area involved in twelve instances did not reach beyond the posterior commissure, whereas in the typical cases, 19 in all, the leucoplakia extended from the mons veneris to the peri-anal skin inclusive.

Age.—The accompanying table clearly shows leucoplakic vulvitis is essentially a disease of old age.

TABLE

20–30	30–40	40–50	50–60	60–70	70–80	80
1	5	3	8	14	9	1

Sixty per cent of the women were over sixty years old. Of the six who were under forty, four merely had patches of leucoplakic vulvitis and one was an artificial menopause after a hysterectomy. That leaves but one case out of forty in which a typical complete leucoplakic vulvitis developed in a woman before the menopause period.

All but nine of the women had had one or more children, five of them were nullipara, and four were virgins.

Pruritus.—In only one patient was pruritus absent and in a few others it was only mild. As a rule, however, it was constant and unbearably severe. In 6 instances the pruritus had existed for less

than a year before the patient consulted me. In 20 pruritus had been noticed for from one to four years, 18 had had pruritus from five to 30 years, and in the remaining case no satisfactory answer could be obtained. As a rule, the pruritus became increasingly severe as the disease advanced though there were often long intervals in which this symptom was less pronounced, sometimes as a result of treatment

Fig. 8.—Leukoplakic vulvitis (first stage). Typical symmetrical involvement from mons to peri-anal region.

and sometimes without any treatment whatsoever. This tendency to recurrence, after temporary relief by local measures or corpus luteum has been an added reason for favoring surgical measures in the treatment of these cases.

Gross and Microscopic Pathology.—Although leucoplakic vulvitis is a slowly progressive disease, we can, I believe, distinguish three

stages, merging one with the other. In the first stage the vulva assumes a swollen reddened appearance, at times having a moist surface and showing only here and there whitish or pinkish-white areas (Fig. 8). Microscopically at this time we find pronounced acanthosis with slight increase of the keratin layer in the epidermis while in the connective tissue there is marked round cell infiltration with occasional leucocytes especially in the papillary spaces. In the second stage of the disease, appearing usually not until about one year has elapsed we find a markedly thickened, white skin with stiff edges and often excoriated from scratching. Microscopically we see pronounced

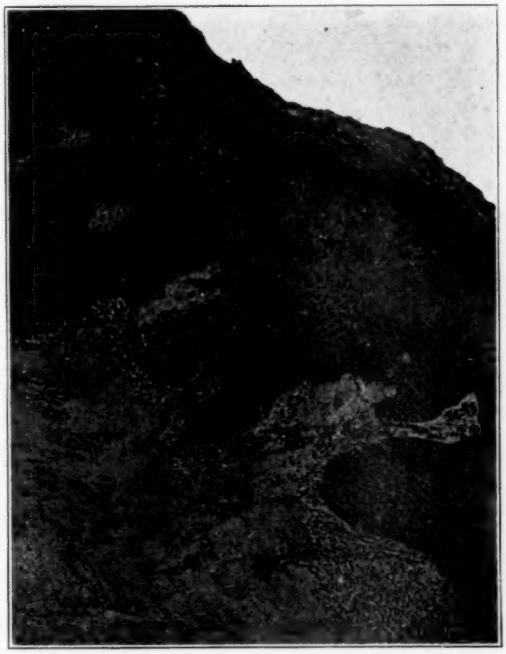

Fig. 9.—Leukoplakic vulvitis (second stage). Marked hyperkeratosis, increase of eleidin cells, acanthosis, round cell infiltration and collagenic changes in the dermis.

changes in the epidermis. The acanthosis is still marked but there is also a piling up of the keratin layer and a thick deeply staining layer of eleidin cells. The connective tissue shows the picture of chronic inflammation with here and there a collagenous appearance. Numerous plasma cells are present (Fig. 9).

In the third stage the skin has assumed the appearance of thin parchment, pearly white, crackled and dry. The epithelial layer is low, papillary processes are absent, the keratin layer usually a half or a third as thick as before, the eleidin cells less numerous and a peculiar frayed out appearance of the basal cells of the epidermis. In the connective tissue we see a glairy looking collagenous material

replacing the connective tissue, with here and there islands of round cell infiltration.

Characteristic of all three stages is the absence of elastic tissue in the subepithelial layers of the dermis. This absence of elastic fibers becomes increasingly marked as the disease progresses.

It is this absence of elastic tissue that I believe is primarily responsible for the disease. My explanation of its etiology would be as follows. In some women at or after the menopause or through some special disturbance of ovarian secretion there occurs a complete disappearance of elastic tissue in the upper layers of the vulval skin about the introitus. This loss of elasticity leads to greater friability, so that minute breaks of continuity readily occur. Through such cracks bacteria gain entrance to the dermis and produce a low grade inflammation. The exudate thus resulting causes pruritus. This, in turn, through the traumatism of rubbing or scratching, produces more cracks and more infection, and so the vicious circle progresses until the characteristic changes above described have been produced.

Cancer may develop at any of the three stages, but is most frequent and most malignant when it originates in the second stage, that of epithelial hypertrophy. In the third stage if a cancer appears, it is often everting in type, slow in growth and runs a relatively benign course.

3519 WASHINGTON AVE. (*For discussion, see p.* 495.)

THE PREVENTION OF FETAL ASPHYXIA*

BY R. A. BARTHOLOMEW, M.D., F.A.C.S., ATLANTA, GA.

THE physician, engaged to take charge of an obstetric patient throughout pregnancy, assumes a responsibility distinctly greater than that pertaining to any other type of practice. He becomes responsible for two patients and accordingly must bear in mind, at all times throughout pregnancy and labor, that the purpose and object in view is not alone to safely relieve the mother of her burden in due time and restore her to normal activity, but to achieve the end and object of the whole process—that the child shall be born alive and with as good prospects of development as it is scientifically possible to attain.

An increasing sense of this dual responsibility has found its expression in prenatal care, whereby the mother, and through her, the child, are given closer supervision throughout the period of intrauterine development. However, a discussion of what constitutes efficient prenatal care and the benefits that are to be derived from it

*Read at the sixty-eighth Reunion of the Alumni of Emory University School of Medicine, Atlanta, Ga., June 5, 1923.

for both mother and child, would amplify this paper far beyond the scope of its title, and it is rather my purpose to emphasize those factors which predispose to asphyxia of the child during labor, and stress the diagnosis of threatened asphyxia and the means of prevention.

Asphyxia of the child during labor is unquestionably one of the greatest causes of stillborn but otherwise healthy appearing infants, and it is likewise true that in many instances, the gravity of the complication predisposing to or causing the asphyxia, is such that it cannot always be prevented.

A consideration of the various etiological factors causing asphyxia of the child during labor, is necessary to a proper understanding of the possibilities of prevention. The child, during intrauterine life, may be said to breathe through the lungs of its mother, through the medium of her blood. The oxygen and carbon dioxide as well as other chemical products of waste and nutrition pass through the thin walls of the villi of the placenta. The respiratory center in the medulla of the fetus normally does not stimulate breathing efforts until at the moment of birth when contraction and retraction of the uterus separate the placenta and so diminish the maternal blood flow to the placental site that the carbon dioxide content of the fetal blood is rapidly increased. There may also be some stimulation of the respiratory center from the chilling of the body surface and the external stimulation that takes place at birth, but the principal factor is the increase in concentration of carbon dioxide in the fetal circulation.

In general, therefore, the cause of asphyxia of the child during labor is an increase in carbon dioxide content of the fetal blood with resulting stimulation of the respiratory center and premature efforts at respiration.

The causes leading to an increase in carbon dioxide concentration in the fetal blood may be classified as maternal and fetal. Among the maternal causes are those affecting the character of the mother's blood or limiting the supply of blood to the placental area, as, for example, pneumonia or far advanced tuberculosis whereby there is an insufficient absorption of oxygen and elimination of carbon dioxide, which automatically increase the carbon dioxide content of the fetal blood. Cardiac decompensation may have the same effect on account of a failing circulation; likewise an advanced state of anemia or poisons in the maternal circulation as uremic poisoning, drugs or anesthetic agents. Too frequent, too prolonged or tetanic contractions of the uterus may result in such a stagnation of the maternal circulation at the placental site through the hemostatic action of the contracted uterine muscle, that proper interchange of oxygen and carbon dioxide cannot take place and asphyxia results.

The injudicious use of pituitrin accounts for many cases of asphyxia during labor.

Among the fetal causes are those affecting the placenta and the fetal circulation through the cord. Premature separation of a normally implanted placenta, placenta previa, direct compression of a low-lying placenta by the head, pathologic changes in the placenta as a result of extensive infarcts or syphilitic infection interrupt the exchange of oxygen and carbon dioxide and lead to asphyxia, either gradually or suddenly. Direct pressure on, or constriction of the umbilical cord by knots, coils, twists or prolapse is a frequent cause. Finally, the external pressure on the head itself by a tight, contracted pelvis during labor, or severe or prolonged compression of the head by forceps, or internal compression of the medullary centers by intracranial hemorrhage, may produce asphyxia of the child during labor.

The diagnosis of threatened asphyxia of the child during labor can be made if the physician follows the fetal heart sounds by frequent auscultation. A gradually increasing concentration of the carbon dioxide in the fetal circulation, brought about by one or more of the various etiological factors previously mentioned, stimulates the vagus inhibitory center of the heart, in the medulla and produces a slowing of the fetal heart rate for some time before the stage of respiratory center stimulation and asphyxia is reached.

The normal range of the fetal heart rate during labor is from 120 to 160. It is not unusual to find the heart rate temporarily accelerated from 160 to 200 or temporarily slowed from 120 to 100. However, a heart rate, either temporarily or persistently accelerated above 160 does not, in my opinion, have the serious significance during labor which a temporary or persistent slowing below 120 indicates.

It is unfortunate that the diagnosis of threatened asphyxia is based, in many textbooks and in some of the medical literature, both on abnormally slow and unusually rapid fetal heart tones. It is confusing and misleading to attempt to harmonize these conflicting statements. Theoretically, the vagus center, after a long continued primary stimulation from an increased carbon dioxide content in the fetal blood, becomes paralyzed with the result that the previously slow heart sounds become abnormally rapid (180 to 200 or above). Practically, however, such an effect I have never seen. The simplest form of temporary stimulation of the vagus, with resulting slowing of the fetal heart rate to perhaps 110 or 100, is found during the labor pain, as a result of the temporary stagnation of the maternal circulation at the placental site by the contracting musculature of the uterus. However, this temporary slowing is immediately followed by a more rapid, normal rate, as soon as the uterus relaxes. If the etiological factor is of such a nature that the carbon dioxide content of the

fetal blood is gradually and continually increased, it is noticed that the fetal heart sounds do not recover their normal rate between pains, but tend to become persistently slowed, below 100, and may be found as low as 40 or 50 per minute. The slowing continues to be more marked during than between contractions.

As the tendency to asphyxia increases, there is apparently a stimulation of the intrinsic nerve mechanism of the intestinal musculature, which results in stimulation of intestinal peristalsis and expulsion of meconium in the amniotic fluid. The appearance of greenish or dark meconium-stained amniotic fluid during labor with a vertex presentation, practically always indicates a tendency to asphyxia, but is almost always a physiological phenomenon and of no pathological significance in breech presentation.

After a period of continued persistent slowing of the fetal heart, the concentration of carbon dioxide and lack of oxygen stimulate the more resistant respiratory center and the infant begins to make respiratory efforts, which become spasmodic and accompanied by violent convulsive movements if air is not obtained. These spasmodic violent movements may frequently be seen and interpreted, if the head is beginning to crown.

If the birth of the baby does not occur until asphyxia is almost complete, there may be such a severe degree of injury to the medullary centers that all means of resuscitation prove to be of no avail. The heart may be made to increase from a very slow, weak, almost imperceptible impulse to rapid, strong pulsations by administering oxygen through mouth-to-mouth breathing or by means of an oxygen tank. In many instances, however, irreparable damage has been done to the vital nerve centers and the infant makes no voluntary effort to breathe. In these cases, it has been my observation, that the heart rate invariably becomes slower and slower and death of the infant occurs at the end of a series of very slow, weak pulsations and is not associated with a terminal, very rapid heart rate.

I would, therefore, particularly emphasize the fact that rapid, fetal heart tones are of no pathological importance in the diagnosis of either a threatened or an advanced state of asphyxia of the child during labor, but that slowing of the fetal heart tones below 120 is of the greatest significance.

Auscultation of the fetal heart sounds during labor has been greatly facilitated in recent years by the use of the De Lee head stethoscope, which represents a most valuable addition to the obstetrician's equipment. The principal advantage of this instrument over the ordinary Bowles or Ford model, for obstetrical use, are found in its adaptation to be held on the head ready for use at any time even though the operator may be "scrubbed up," and also in its increased power of

conducting the fetal heart sounds, probably as a result of some bone conduction through its rigid metal construction.

A modification of the De Lee head stethoscope has been made by G. P. Pilling and Son Co., of Philadelphia, following a suggestion that a joint, placed between the end piece and the head piece, would permit a better adjustment of the end piece to suit different locations of the fetal heart sounds, and still not impair the distinct transmission of the sound. In listening to the heart sounds when the back of the fetus is toward the flank, one finds, with the rigid connection, that it is necessary to stoop and extend his head into an awkward position, to hear the heart sounds, and if he wishes to auscultate on the opposite side of the abdomen he must either go around to the other side of the bed or else turn the patient on her side, toward

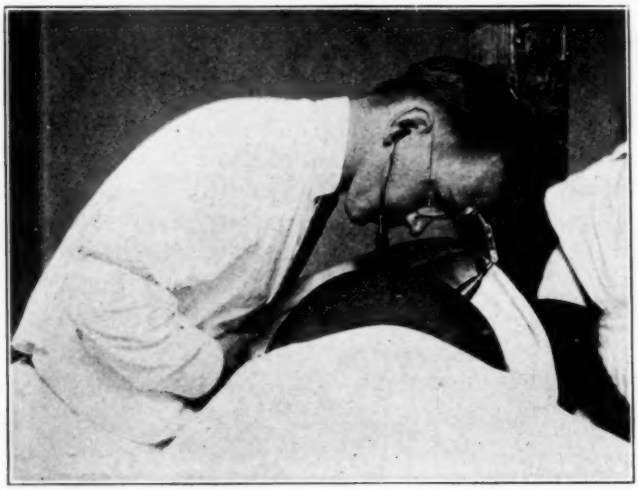

Fig. 1.—Use of the modified DeLee head stethoscope adjusted for auscultation on the far side of the abdomen.

him. The adjustable joint, however, enables one to change the direction of the end piece and auscultate over any portion of the abdomen with ease and with no diminution in sound transmission. The rubber tubing if too short, or too stiff, does not allow a sufficiently free excursion of the end piece and may strain the ears. It should be of a soft, flexible quality, preferably handwrapped stethoscope tubing, lumen 4 mm. in diameter, wall 2.5 mm. in thickness and each piece should measure 7 inches in length.

During the first stage of labor the fetal heart sounds should be followed every hour or oftener if possible, and if there is any slowing noticed, particularly between pains, so that the rate falls to between 100 and 120, auscultation should be continued over a period of several labor pains, both during and between the contractions to determine whether the slowing is merely temporary or tends to be persistent. A temporary slowing of this degree (120-100) is often

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noticed during a uterine contraction, but if due solely to this, the rate increases promptly to normal limits between contractions. If there is some more serious factor at fault, the fetal heart rate may increase slightly between pains but fails to come back to the lower limit of normal (120).

It then becomes necessary to follow the fetal heart sounds at close intervals and discover the cause if possible. An examination bearing upon the various possible factors, may indicate the reason for the threatened asphyxia and point to the proper treatment. If the cause can be removed, this should be done. If it is not possible to remove the cause or deliver the patient promptly, the life of the fetus may be prolonged by administering oxygen to the mother as recommended in Shears' Obstetrics, until effective treatment can be carried out. Too rapid and prolonged uterine contractions may require the temporary administration of a small amount of anesthetic to relax the uterus and re-establish circulation at the placental site. Pituitrin should not be given without first ascertaining whether the fetal heart rate indicates any tendency to asphyxia and it is not wise to give it unless the dilatation and progress have reached such a stage that the child could be promptly delivered if necessary. The treatment indicated will depend not only on the nature of the factor producing asphyxia, but also on the stage of labor. In some cases in proper surroundings cesarean section may be indicated, in other cases version or forceps.

As to an overdosage of drugs such as morphine, hyoscine or anesthetic agents, it has seemed to me that there is no noticeable effect on the infant's heart rate while it is still in the uterus and dependent on the maternal circulation, but at birth the depressing action of the absorbed drug on the infant's respiratory center may be so marked that respiration may be markedly delayed or fail to be established. Such effects are apt to be seen in "twilight sleep" babies. It is not advisable to give such drugs at any time within several hours preceding the time at which the child will probably be born.

During the second stage of labor, particularly when the head reaches the perineum, the cord, which is so frequently around the neck one or more times, tends to be compressed between the back of the neck and the posterior surface of the pubic bone. This is the stage of labor during which the physician should auscultate the fetal heart sounds at intervals of 10 to 15 minutes in order to discover evidence of pressure on the cord brought about in this manner. Furthermore, in every labor it is always best to have the forceps sterilized and ready to use if necessary, from the beginning of the second stage. If this has not already been done, they should be prepared at once if the heart rate shows a tendency to remain persistently from 120 to 100 per minute. If the heart rate becomes less

than 100 the patient should be brought to the edge of the table or bed, in position for forceps delivery and the anesthetic should be in readiness to be used at once if necessary. If the heart rate persists below 100 and tends to go still lower, a forceps delivery should be done without delay. These precautions and measures of preparedness may save the infant's life. Failure to have the forceps ready and the patient in position ready to be anesthetized at once, may result in failure to save the infant, even though the diagnosis may have been made by discovering a slowing heart rate. The necessity for immediate forceps delivery becomes more urgent if, in addition to abnormally slow heart sounds, the partially crowned head of the baby is seen to extend in sudden, spasmodic motion, irregularly or rhythmically, indicating violent efforts on the part of the baby to obtain air. Meconium stained fluid is not likely to be seen at this stage due to the fact that the head completely blocks the canal from above. If forceps are not at hand or prepared, immediate anesthesia induced by rapid deep breathing, and delivery by episiotomy and forcible pressure on the fundus is indicated. Pituitrin is of doubtful use in such an emergency as its action is not sufficiently prompt and, furthermore, might increase the asphyxia.

Auscultation of the fetal heart sounds late in the second stage of labor, when the head has reached the perineum, will frequently show a moderately slow rate, varying from 120 to 100, probably due to the increased pressure of the close pelvic outlet on the baby's head, the more frequent and prolonged contractions preceding birth, the bearing down efforts of the mother, and the beginning diminution of the placental site from retraction and contraction of the muscle of the fundus. One should not interfere hastily, on noting this moderate degree of slowing of the heart rate (120 to 100), but should continue auscultation at 5 to 10 minute intervals, meanwhile making preparations to interfere should the rate persist below 100, with tendency to become slower and slower.

The following abstracts of cases taken from personal records illustrate the practical value of a close observation of the fetal heart sounds during labor.

CASE 1.—Mrs. P. C., primigravida, went into labor at full term, 4 A. M., March 11, 1922, following a normal, uneventful pregnancy. Her general physical condition was good, and the pelvic measurements were normal. The presentation was O.R.A. The uterine contractions occurred at five minute intervals, gradually lessening to two or three minutes and increasing in severity during the forenoon. The membranes ruptured at 2 P. M., and the fluid at that time showed no meconium. Auscultation of the fetal heart at 7 A. M., showed it to be 120 per minute, strong and regular. However, at 10 A. M., it was noted that the rate diminished to 60 per minute during the pains but returned quite promptly to 120-140 between pains. A vaginal examination showed the cervix dilated about 3 cm., and no presentation or prolapse of the cord in advance of the head, which had by this time, descended

to just above the ischial spines. The patient had been taking nitrous-oxide-oxygen analgesia during the preceding half hour, with considerable relief. There seemed to be no factor to account for the slow heart rate during pains, other than possibly a coil of cord around the neck or else a loop of cord low enough to be pressed against the lateral pelvic wall. At 2 P. M., a vaginal examination showed the cervix fully dilated and the head low and completely rotated. Up to this time, frequent auscultation of the heart sounds showed the same characteristics—a decrease in rate to 60 during pains, but a fairly prompt recovery to 120 to 130 between pains. The forceps were sterilized and the patient prepared for delivery, should interference become necessary. At 2:30 P. M., there was a definite failure of the heart sounds to recover their normal rate between pains and within another half hour the heart rate remained below 100 both during and between pains. A low forceps delivery was done at once under ether anesthesia, and following the birth of the head there escaped a large amount of meconium-stained amniotic fluid, which had previously been held back by the head. A single coil of cord was around the neck. The baby was born at 3:10 P. M., and attempted to breathe before delivery of the shoulders. The cry was somewhat delayed but no active measures of resuscitation were necessary. The cord presented a very unusual condition in that there was an almost total lack of Wharton's jelly, leaving the large umbilical vein and arteries entirely unprotected and consequently very subject to compression between the neck and the pubic bone.

CASE 2.—Mrs. A. K., primigravida, went into labor at full term Sept. 14, 1922, about 10 P. M. There had been no complications during pregnancy and her general physical condition and pelvic measurements were normal. The presentation was O. L. P. Throughout the first stage the fetal heart sounds remained normal 130 to 140, and the uterine contractions occurred at 3 minute intervals but were not very severe. The membranes ruptured spontaneously at 12:30 P. M., Sept. 15, and there was no meconium in the discharge. At 1:30 P. M., a rectal examination showed the cervix fully dilated, the head on the perineum and the occiput anterior. Nitrous-oxide-oxygen analgesia was given from 12 noon almost up to the time of delivery. On account of inefficiency of the pains and failure of the slightly crowned head to advance, 3 minims of pituitrin were given at 2:10 P. M., but with very little effect on the contractions. Auscultation of the fetal heart at 2:20 P. M., showed a marked slowing of the rate to 70, which up to that time had been 130 to 140 on repeated counts. Preparations were made to interfere quickly if necessary. The heart rate which for a time tended to increase somewhat between pains, soon persisted at a low rate between as well as during the pains and at 2:35 P. M., the partially crowned head was seen to make sudden, spasmodic movements as though the baby was trying to breathe. The forceps were immediately applied, with the patient under nitrous-oxide-oxygen anesthesia, an episiotomy was done, and the baby delivered at 2:40 P. M. On delivery of the head, there was found a loop of cord prolapsed alongside of the body and neck. The baby was a 7¾ pound male infant, and born in a condition of pallid asphyxia. The heart beat was about 36 per minute and very weak. Simple methods of resuscitation were of no apparent value, but after about 15 minutes of mouth to mouth artificial respiration, the baby began to breathe and was soon in fairly good condition, and continued to be normal.

SUMMARY

The double responsibility of obstetric practice demands that the physician give due regard to safeguarding the life of the child during labor as well as during pregnancy. Death of the child during

labor is practically always due to asphyxia, brought about by various maternal and fetal causes, some of which act slowly and are preventable, while others are so rapid in their action that little opportunity is given for treatment.

A lack of oxygen and an increase in carbon dioxide in the fetal blood causes a stimulation, first, of the cardio-inhibitory center in the medulla, which results in a decrease of the fetal heart rate. Later on, the respiratory center is stimulated, and with ineffectual efforts to breathe, asphyxia results. A fetal heart rate temporarily slowed, not lower than 100, during pains, but increasing to normal rate between pains, is a sufficient indication to follow the heart rate more frequently, discover the cause if possible, and make preparations for interference, if required. A fetal heart rate persistently below 100, both during and between pains, demands prompt delivery of the baby if this can be done without great risk to the mother. A rapid heart rate does not indicate either a threatened, or an advanced state of asphyxia.

The diagnosis of threatened fetal asphyxia is facilitated by use of the De Lee head stethoscope. The adjustable model made by Pilling & Co., is better adapted to all positions. It should be used at frequent intervals during the second stage of labor, when compression of the cord, coiled about the neck or partially prolapsed, against the pubic bone, occasionally produces asphyxia. Prompt delivery of the baby, under such circumstances, may prevent death from asphyxia.

20 PONCE DE LEON AVENUE.

THE CALCIUM AND MAGNESIUM CONTENT OF THE BLOOD SERUM DURING PREGNANCY, LABOR AND THE PUERPERIUM

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WE have recently been led to undertake the study of certain of the more obscure problems of the physiology of gestation in order better to attack the subject of the toxemias of pregnancy and, with this in mind, have examined the calcium content of the blood serum of 205 normal nonpregnant, pregnant, parturient and puerperal women to determine the usual physiological variations incident to gestation. Whenever possible, we have also made observations upon patients suffering from the toxemias of pregnancy. During one period of the study a considerable number of magnesium determinations were made, but when no very striking variations from normal were found, this work was largely abandoned.

Among the many theories which have been advanced to explain the occurrence of eclampsia, is that of calcium deprivation of the mother incident to her need for supplying large quantities of this mineral to the various tissues of the developing fetus. The magnitude of this demand has been determined through the analysis of fetuses at the different months of intrauterine development by Michel,[1] Camerer, Söldner and Herzog,[2] Givens and Macy,[3] and others. These observers all agree that a full term fetus contains from 20 to 30 grams of calcium and approximately 1 gram of magnesium. The maternal organism must, therefore, have an average positive balance during pregnancy of approximately 100 mg. calcium and 4 mg. magnesium per day, if the fetal demands are to be met without any withdrawal of the body's fixed reserves.

The inorganic needs of the fetus are practically insignificant until about the middle of pregnancy, but from that time progressively more calcium and magnesium are necessary for fetal growth. Consequently, the danger of a real deprivation is greater as term is approached, and would in fact seem inevitable unless the maternal metabolism were conducted more economically than at other times, or unless the diet provided an unusual quantity of these elements. We have succeeded in finding only one calcium and magnesium balance experiment in the available literature, that of Hoffström[4] published in 1910. This author studied one normal patient from the seventeenth week of pregnancy to the time of delivery and concluded that the metabolism of

the organism so adapted itself to the altered requirements of gestation that, even upon an ordinary diet, there was no sacrifice entailed by the necessity for supplying the building stones for the fetus. In the case of all the elements studied—nitrogen, sulphur, phosphorus, calcium and magnesium—a positive balance obtained almost from the beginning of the experimental period and the storage became more pronounced as term was approached. In consequence of this economy, the ingested food not only supplied all the materials needed for development of the product of conception, and the coïncidental hypertrophy of the uterus and breasts, but in each instance there was still a positive balance accruing to the rest of the body, supposedly in anticipation of the demands of the lactation period. Improved assimilation and diminished excretion apparently play the leading rôles in this adaptation.

This work certainly argues against a physiological mineral deprivation of the organism as a whole during pregnancy, but does not actually disprove the idea that a lowered blood calcium may be a part to its various pathological manifestations. Moreover, the demonstrated association of diminished blood calcium with tetany and the fancied resemblance of this disease to eclampsia, together with the efficiency of calcium therapy in the treatment of the former, have revived the theory and stimulated many workers. Within the past few years several authors, including Drennan,[5] Oliver,[6] Halford,[7] Oettingen,[8] Loew,[9] and others have advocated the hypothesis upon purely theoretical grounds, or upon the clinical results of calcium administration or dietary regulation. The latest of these, Oettingen,[8] has most attractively combined a supposed calcium deficiency with Zangemeister's *hydrops gravidarum* theory[10] and Fischer's conception of edema[11] to explain the disease upon the basis of physico-chemical changes, but has failed to advance any proof of his contention.

On the basis of this hypothesis, attempts have likewise been made to produce eclampsia-like conditions in experimental animals by reducing the blood calcium by means of chemical agents. Burckhardt-Socin[12] lowered the calcium in the circulating fluid by intravenous injections of oxalate radicals and described clinical disturbances which bore some resemblance to eclamptic manifestations. This work has not been confirmed, and, moreover, the mere demonstration of such results is of little value unless similar alterations in blood calcium are found in puerperal eclampsia to substantiate the conclusions, which can be so easily drawn from laboratory experience alone.

NORMAL VARIATIONS IN SERUM CALCIUM AND MAGNESIUM DURING THE REPRODUCTIVE CYCLE

Actual analyses of the blood for calcium (and magnesium) during normal pregnancy have been reported during the past few years, but

the results are too divergent to be conclusive. The older analytical procedures either required such large quantities of blood as to make repeated determinations impracticable (ashing, precipitation of the calcium as oxalate, and filtration, followed by permanganate titration or weighing as calcium oxide), or gave such varying results that their accuracy must be suspected (Blair-Bell's method of counting the calcium oxalate crystals in diluted blood after the addition of an oxalate solution).

With these methods the results were, for the most part, unsatisfactory. Lamers[13] reported an increased plasma calcium during pregnancy with a considerable further rise at the time of labor, and a postpartum drop to the pregnancy level, where the values remained during lactation. Kehrer[14] in 1913 stated that in normal pregnancy he had found the whole blood calcium somewhat increased, but that blood samples taken nine or ten days after delivery showed a considerable decrease. He attributed the puerperal diminution to blood lost at the time of labor, to lactation and to a calcium poor diet. Linzenmeier[15] and Aymerich[16] in the same year, confirmed Kehrer's finding with regard to the calcium increase in whole blood during the second half of pregnancy, but made no determinations upon puerperal patients. Jansen[17] found practically no change in the whole blood calcium during any part of the reproductive cycle, although the tendency was toward an irregular increase. Meigs, Blatherwick and Cary[18] were likewise unable to detect any variations in the calcium content of the plasma of cows during pregnancy or lactation. On the other hand, Morley,[19] using the Blair-Bell technic in 1913, concluded that "* * * there is a lessened amount of calcium in the blood of pregnant women and of women in labor."

Kehrer,[20] in 1920, repeated his earlier work upon whole blood and came to quite different conclusions, which were later confirmed by Mazzocco and Moron.[21] Kehrer found that the values during late pregnancy, at the time of labor, and in the early lactation period are generally somewhat below normal, although not infrequently normal concentrations obtain. He says that, "One may, therefore, speak of a low degree of physiological deprivation in the organism at the end of pregnancy and in the early puerperium * * *"; and, furthermore, offers three possible explanations for the observed changes: (1) the physico-chemical properties of the blood become altered so that the calcium salts are less readily soluble than in the nonpregnant state, (2) the blood becomes diluted, (3) calcium assimilation is retarded in some unknown way.

Since it is known that the corpuscles contain practically no calcium, it would seem that the analyses of serum or plasma would give more useful information, particularly when it is remembered that

the percentage volume of cells in whole blood is subject to marked physiological variations. It seems probable that little or no calcium is used up in the process of clotting and that the calcium content of plasma and serum is, therefore, practically identical. Following the appearance within the past four years of more accurate methods of analysis upon small samples of serum or plasma, certain other publications upon the subject have appeared, the majority of them since this work was begun.

Hess and Matzner[22] determined the serum calcium by Lyman's method and in 11 antepartum specimens found an average of 10.4 mg. per 100 c.c. while in 18 early postpartum samples the average was 9.75 mg. per 100 c.c. Since no normal control figures are given, it is impossible to say whether there is an appreciable change attributable to pregnancy, although a comparison with the generally accepted normal values would indicate that there is no significant alteration. On the other hand, their figures show a relative diminution in the first 48 hours after delivery, even though the absolute quantities are open to criticism by reason of the demonstrated inaccuracies of the method employed.

De Wesselow,[23] using the Kramer-Tisdall method,[24] examined the sera of 17 normal nonpregnant women, 32 normal pregnant women from the third to the ninth month of gestation, and 22 puerperal patients (9 early and 13 late). The average values during pregnancy were all slightly lower than in the nonpregnant controls, indicating a definitely lower trend for the serum calcium during this period, but only in the six-to-seven-months period was the average below the normal limits, and in every group there were samples in which the calcium was quite normal. In both the early and late puerperium, the calcium values were normal, but it should be noted that no specimens were obtained during the first two days after delivery.

Krebs and Briggs[25] recently reported some studies of the organic and inorganic blood constituents in 17 normal pregnant women and conclude as follows: "The results * * * show a great constancy of all the elements in normal pregnancy, regardless of the period of gestation, with one exception, namely, that in the last weeks of pregnancy our few cases showed the calcium content to be slightly lowered." They found the calcium of the plasma or serum to vary from 7.5 to 10.0 mg. per 100 c.c., while the magnesium ranged from 2.1 to 2.7 mg. per 100 c.c.

Leicher,[26] in 3 cases of normal pregnancy at the third and fourth months, found the serum calcium to be normal, while among 10 women nearly at full term 5 showed normal values, 2 were in the lower range of normal and 3 were distinctly low, and in 9 puerperal patients 6 were normal and 3 were subnormal. These values must

be considered only as comparable within themselves, since the absolute values are considerably higher than those usually accepted as normal.

There is thus seen to be a certain divergence of opinion as regards the normal variations of blood calcium during reproduction and, moreover, the individual authors have reported series of cases altogether too small to permit of general deductions, or have employed analytical methods which are open to criticism, while magnesium determinations seem not to have been attempted except by Krebs and Briggs.[25]

Under these circumstances, it seemed advisable for us to undertake a more extended series of observations upon the calcium and magnesium content of the serum during normal pregnancy, at the con-

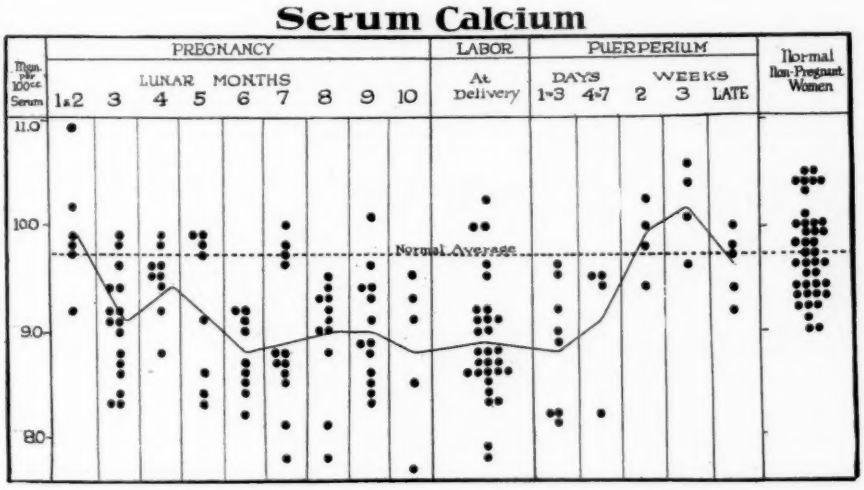

Fig. 1.

clusion of labor, and at various periods after delivery. We have, therefore, determined the serum calcium in 40 normal nonpregnant women controls, 105 normal pregnant women fairly evenly distributed from the fifth to the fortieth week of pregnancy, 29 women at delivery and 31 from twelve hours to twelve weeks postpartum; and the serum magnesium in 16 normal nonpregnant women in the childbearing age, 29 during pregnancy, 23 at the end of labor and 18 during the postpartum period.

EXPERIMENTAL

The blood samples were collected from an arm vein and allowed to clot, after which the clear serum was separated by centrifugalization. The calcium was determined by the direct precipitation method of DeWaard[27] as modified by Kramer and Tisdall,[24] while

the magnesium determinations were made by a combination method recently described by us,[28] which gives values comparable to those of the procedures generally employed. All determinations were made in triplicate and every precaution was taken to insure their accuracy.

The calcium values obtained are presented in Table I, while Figure 1 is a graphic representation of the same data in which individual determinations are represented by dots, in order that the occurrence of exceptional cases may not be lost sight of, as so often happens when only average values are recorded. The continuous line connects the average values in each period and shows the general trend of the concentration of calcium in the serum during the various stages of the child-bearing period. The figures for healthy nonpregnant women are introduced for comparison, and in Fig. 1 the dotted horizontal indicates the average of these forty determinations.

TABLE I

SERUM CALCIUM IN NORMAL PREGNANCY, LABOR AND PUERPERIUM

SUBJECTS	NUMBER OF DETERMINATIONS	SERUM CALCIUM	
		AVERAGE	RANGE
		mg. per 100 c.c.	mg. per 100 c.c.
Normal nonpregnant women	40	9.7	9.0-10.6
Pregnancy			
1- 8 weeks	7	9.9	9.2-10.9
9-12 "	19	9.1	8.3- 9.9
13-16 "	12	9.4	8.8- 9.9
17-20 "	11	9.1	8.3- 9.9
21-24 "	11	8.8	8.2- 9.2
25-28 "	15	8.9	7.8-10.0
29-32 "	11	9.0	7.8- 9.5
33-36 "	14	9.0	8.3-10.1
36-40 "	5	8.8	7.7- 9.5
At the time of delivery	29	8.9	7.8-10.3
Postpartum			
1- 3 days	8	8.8	8.1- 9.6
4- 7 "	7	9.1	8.2- 9.5
8-14 "	7	9.7	9.4-10.3
15-21 "	4	10.2	9.6-10.7
more than 21 days	5	9.6	9.2-10.0

It should be noted that after the second month of pregnancy the serum calcium tends to be somewhat lowered, although occasionally it may even be slightly above the normal average. As term is approached these exceptions become less numerous and are quite unusual at the time of labor. During the last five lunar months of pregnancy the average serum calcium values never exceed the lower limit for normal as found in our series of nonpregnant women (9.0 mg. per 100 c.c.), and are consistently maintained at a level considerably lower than the average for normal nonpregnant women.

At the end of labor and during the first three days of the puer-

TABLE II

NORMAL SERUM MAGNESIUM VALUES

SUBJECTS	NUMBER OF SPECIMENS	AVERAGE VALUE	RANGE OF VALUES	INDIVIDUAL DETERMINATIONS
		mg. per 100 c.c.	mg. per 100 c.c.	mg. per 100 c.c.
Healthy nonpregnant women	16	2.29	1.9-2.7	1.9 2.0 2.0 2.1 2.1 2.2 2.2 2.2 2.2 2.3 2.3 2.4 2.6 2.6 2.7 2.7
Pregnant women	29	2.26	1.5-2.9	
1- 8 weeks	4	2.35	1.6-2.9	1.6 2.2 2.7 2.9
9-12 "	5	2.04	1.5-2.4	1.5 2.0 2.1 2.2 2.4
13-16 "	3	2.13	1.9-2.5	1.9 2.0 2.5
17-20 "	2	2.50	2.1-2.9	2.1 2.9
21-24 "	2	2.45	2.4-2.5	2.4 2.5
25-28 "	4	2.33	2.0-2.7	2.0 2.1 2.5 2.7
29-32 "	5	2.38	2.1-2.7	2.1 2.3 2.3 2.5 2.7
33-36 "	3	2.10	2.0-2.2	2.0 2.1 2.2
37-40 "	1	2.10	2.1	2.1
At the end of labor	23	2.03	1.4-3.2	1.4 1.6 1.6 1.7 1.7 1.7 1.7 1.8 1.8 1.9 1.9 1.9 1.9 1.9 2.0 2.1 2.2 2.3 2.4 2.6 2.6 2.8 3.2
Puerperal women	18	2.16	1.7-3.0	
1- 3 days	3	2.03	1.7-2.6	1.7 1.8 2.6
4- 7 "	2	1.80	1.8	1.8 1.8
8-14 "	4	2.43	2.0-3.0	2.0 2.2 2.5 3.0
15-21 "	5	2.26	1.9-2.8	1.9 2.0 2.2 2.4 2.8
more than 21 days	4	1.98	1.8-2.2	1.8 1.9 2.0 2.2

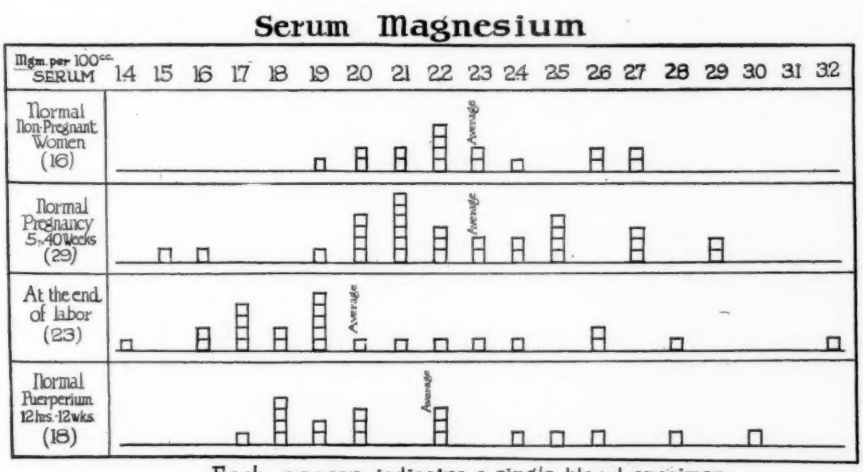
Serum Magnesium
Mgm. per 100cc. SERUM
14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32
Normal Non-Pregnant Women (16)
Normal Pregnancy 5-40 Weeks (29)
At the end of labor (23)
Normal Puerperium 12 hrs.-12 wks. (18)
Average
Each square indicates a single blood specimen.

Fig. 2.

perium, the average values for serum calcium are very similar to those found in later pregnancy. In individual cases there seems to be a tendency toward a slight increase at the time of labor, followed immediately by a fall, but this is only weakly reflected in the average values. During the latter part of the first week postpartum, there is an appreciable rise in the curve, and a normal serum calcium seems

to have been restored by the second week, the rate of recovery varying with individual patients. Some tendency toward over-compensation may be indicated by the high values obtained during the third week of lactation but the number of cases is too few to be conclusive. It would seem, however, that the continuance of lactation is not associated with a high serum calcium, as evidenced by the normal figures obtained in the late puerperium.

The serum magnesium values (Table II and Fig. 2) obtained upon 70 obstetrical patients ranged from 1.4 to 3.2 mg. per 100 c.c., while the average figures varied from 1.8 mg. per 100 c.c. in the latter part of the first week postpartum to 2.5 mg. in the fifth month of pregnancy, as against 2.3 mg. per 100 c.c. in the 16 normal nonpregnant women. Of the values obtained during pregnancy, 21 of the 29 were between 2.0 and 2.5 mg. per 100 c.c. (Fig. 2), whereas during labor 13 out of 24 were between 1.6 and 1.9 mg. per 100 c.c., with the others scattered over a wide range, and during the puerperium 12 out of 18 were between 1.8 and 2.2 mg. per 100 c.c. Among the 16 normals, 11 lay

TABLE III

NORMAL SERUM CALCIUM AND MAGNESIUM: REPEATED DETERMINATIONS UPON INDIVIDUAL CASES

PATIENT	DATE	PERIOD	CALCIUM	MAGNESIUM
			mg. per 100 c.c.	mg. per 100 c.c.
H.....	1-20-23	36 wks. preg.	9.4	2.2
	2-20-23	At delivery	9.1	1.8
D.....	12-19-22	24 wks. preg.	8.6	..
	4-19-23	9 d. p.p.	9.4	..
C.....	1-13-23	25 wks. preg.	8.8	2.0
	4-19-23	10 d. p.p.	9.4	..
M.....	2-15-23	At delivery	8.5	1.8
	2-17-23	2 d. p.p.	9.6	1.7
H.....	2-14-23	At delivery	8.8	1.7
	2-17-23	3 d. p.p.	8.9	1.8
G.....	1-25-23	At delivery	8.6	1.8
	2-10-23	16 d. p.p.	10.5	2.4
O.....	2- 3-23	At delivery	8.6	1.9
	2-10-23	7 d. p.p.	9.4	1.8
A.....	1- 6-23	At delivery	8.8	1.7
	1-19-23	13 d. p.p.	10.3	2.0
	3-20-23	10 wks. p.p.	9.2	..
C.....	2- 5-23	At delivery	8.7	1.9
	2- 8-23	3 d. p.p.	9.2	2.6
L.....	1- 3-23	At delivery	8.6	2.6
	1-19-23	16 d. p.p.	10.7	1.9
McG....	1-17-23	At delivery	9.0	1.4
	1-27-23	10 d. p.p.	9.8	2.5
C.....	1-15-23	At delivery	8.4	2.4
	1-31-23	16 d. p.p.	9.6	2.8
D.....	1-25-23	At delivery	8.7	2.2
	2-10-23	16 d. p.p.	10.1	2.2

between 2.0 and 2.4 mg. per 100 c.c., figures which agree closely with the few determinations previously reported for normal individuals.

When all the pregnancy magnesium figures are considered together, both the average values and the extremes are seen to correspond very closely with those of the nonpregnant women, and the *mode* is similarly situated. There is perhaps a tendency, in the last two months, for the concentration of magnesium to be slightly diminished but our figures are too few to be conclusive. At the end of labor, on the other hand, the average is distinctly lower (10 per cent) and the *mode* falls at a lower point, while during the puerperium the general tendency is toward more normal values, although the few specimens obtained in the first week after delivery are even lower than those taken at labor. In general, then, it would seem that the serum magnesium varies somewhat as does the calcium and in all probability the same factors are responsible for the observed changes. While the actual variations in the serum magnesium are small, the proportional changes are fairly comparable to those noted for calcium.

In a few instances, repeated determinations upon the same patient showed that the variations in both serum calcium and magnesium, which appeared in average figures, can usually be demonstrated also in the individual (Table III).

DISCUSSION

The interest attaching to such a demonstration of a tendency for the serum calcium and magnesium to be decreased during the latter part of pregnancy, at the time of labor, and during the early puerperium, is largely dependent upon its interpretation. It might be regarded as evidence of a certain degree of depletion of the mineral resources of the maternal organism by reason of the fetal demands, but the experiment of Hoffström[4] offers a fairly conclusive argument against such a hypothesis. Furthermore, we have shown in recently published experiments on the maternal-fetal interchange that the fetal serum calcium is invariably equal to and may exceed the values obtaining in normal adults, while the fetal magnesium is always as high as, and may be higher than, that in the mother's serum, so that presumably the fetus is able to maintain in its circulation the optimum amount of these two minerals needed for its full development, in spite of the lowered concentration of these elements in the maternal serum. In the second place, it might be looked upon as a result of an inefficient assimilation of the ingested mineral food, but again Hoffström's balance experiment would render such a theory very improbable. Finally, it may prove to be merely a relative diminution due to actual dilution of the serum, and we are, at present, inclined to view it in this light.

There is considerable evidence that the blood volume becomes augmented during pregnancy by reason largely of an increased plasma

dilution (hydroplasmia). Miller, Keith and Rowntree[29] demonstrated this in 1915 by their method of dye injection, and all the purely chemical studies available have tended to confirm their opinion. Stander and Tyler[30] found an appreciable increase in the water content of the whole blood and plasma during pregnancy. They maintain that this dilution increases gradually up to the seventh month and subsequently remains stationary or slowly decreases. DeWesselow[23] determined the total solids of the serum during the course of his experimental work and detected a progressive decrease during pregnancy, with a gradual return to normal after delivery. Except during the early days of the puerperium, however, there were occasional specimens which failed to show a diminished solid content. In general, the dilution tended to parallel the calcium values—low solid content being associated with low calcium.

Our own experience, based upon determinations of plasma and serum protein values, to be published shortly, also indicates a gradual dilution, beginning at about the middle of pregnancy and increasing up to full term, with a slight concentration at the time of labor. Following delivery there is a further rapid dilution for the first 48 hours, due apparently to an influx of water from the tissues, but from this point the plasma protein rapidly increases to normal, where it remains during lactation.

Quite usually, an appreciable edema is associated with diminished plasma or serum protein, the diminution being roughly proportional to the accumulation of water in the tissues. During this hydroplasmia, the freezing point depression of the plasma is somewhat decreased, according to Zangemeister,[31] so that a lowered osmotic pressure obtains, although there is an increase in the sodium chloride, which tends to limit the physico-chemical changes produced by the dilution. The antagonistic action of sodium and calcium ions in solution is well established, but it cannot be said with certainty that the variations in calcium concentration are due to such alterations, although such a view is well within reason. Moreover, it cannot, at present, be stated whether the blood dilution (increased water content) is the cause or effect of the inorganic salt changes. At any rate, the lowering of the serum calcium is associated with a dilution of the serum and with an increase of certain other electrolytes, e.g., sodium chloride, so that the total ionic concentration remains very little affected.

CALCIUM AND MAGNESIUM IN THE BLOOD DURING THE TOXEMIAS OF PREGNANCY

Drennan[5] in the course of a series of articles upon the general subject of the abstraction of calcium salts, advanced the theory in 1911 that depletion of the calcium in the maternal organism is the

cause of eclampsia. She failed, however, to adduce any evidence to support such a hypothesis.

Two years previously Halford[7] and Mitchell[32] had suggested the same thing and had reported several patients with toxemia, with and without convulsions, in whom the therapeutic administration of calcium lactate was associated with marked clinical improvement. It would seem, however, that the other measures adopted might in themselves have been responsible for the disappearance of the symptoms. In fact, no conclusive clinical demonstration of the efficacy of even large doses of calcium lactate or calcium chloride in the pregnancy toxemias has yet been forthcoming, and our own experiences have been disappointing.

Oliver[6] (1915) and Loew[9] (1919) suggested that eclampsia may be due to some disturbance of calcium metabolism, and advised a diet rich in milk, vegetables and fruits during pregnancy, in order that a calcium deficiency might be avoided. In this connection, it is certainly not unreasonable to attribute the diminution of eclampsia in certain regions of Central Europe during the war to an increase in the consumption of mineral salts depending upon the dietary changes necessitated by the blockade, rather than to the decreased protein or fat intake, as stressed by the German writers. The beneficial effects of a straight milk diet in the pregnancy toxemias is also more probably referable to the mineral content than to any other factor, although such contentions cannot be accepted as proven facts.

Nowhere have we seen any specific reference to magnesium deficiency as a cause of the toxemias, although Rissmann[33] has advocated the rectal administration of magnesium sulphate solution in toxic patients, in order to reduce the blood pressure, and the intradural injection of the same salt following spinal puncture in eclampsia.

Several authors have recorded scattered calcium analyses of the whole blood, plasma or serum of toxemic women, but only one writer has recorded the magnesium content. Lamers,[13] in 4 cases of eclampsia, found plasma calcium within the range obtaining in his 14 normal subjects. The absolute values (10 to 12 mg. calcium per 100 c.c.) are probably incorrect, but the relation between the various samples suggests that no essential changes appear in eclamptic women. Linzenmeier[15] also found normal values for calcium in the whole blood of 5 eclamptic patients.

Kehrer[14, 20] detected a lowering of the whole blood calcium in both nephritic toxemia and eclampsia, and stated that this condition continues during the puerperium even in spite of an almost exclusive milk diet. In certain instances, however, the values were not below those noted in normal pregnancy, so that no significant conclusions can be drawn.

Mazzocco and Moron,[21] although they found a diminished whole

TABLE IV

SERUM CALCIUM IN THE TOXEMIAS OF PREGNANCY

CASE NO.	MONTH OF PREGNANCY	PARA	SYSTOLIC BLOOD PRESSURE	ALBUMINURIA	EDEMA	DATE	SERUM CA	REMARKS
			mm.				mg. per 100 c.c.	
HYPERTENSION								
1	7	0	220	0	sl.	12-27-21	9.2	So-called "idiopathic hypertension."
	"	"	160	0	0	1- 3-22	10.0	After administration of Calcium Lactate.
	8	"	180	0	0	1-17-22	8.4	Several days after Ca therapy was stopped.
	"	"	140	0	0	1-30-22	8.9	Several days after Ca therapy was re-begun.
PRE-ECLAMPTIC TOXEMIA								
2	10	0	130	+	0	1-26-22	7.3	
3	10	0	140	++	slight	1-30-22	9.3	Fetus dead.
4	10	0	195	+++	mod.	2- 3-22	9.2	Recovered completely. No return of sympt.
5	9	0	135	0	slight	2-14-22	7.9	Mild toxemia.
6	10	0	150	+++	0	2-16-22	8.4	Slightly toxic.
7	9	0	150	1.5 G. per 1.	marked	2-24-22	8.9	Twins.
8	9	0	165	4.0 G. per 1.	0	4- 5-22	9.4	Slight visual disturbances.

TABLE IV—Cont'd.

ECLAMPSIA

CASE NO.	MONTH OF PREGNANCY	PARA	SYSTOLIC BLOOD PRESSURE	ALBUMINURIA	EDEMA	DATE	SERUM CA	REMARKS
9	9	0	165	+++	slight	3-17-22	8.8	After 5 convulsions.
	p.p.	–	120	0	0	4- 7-22	9.1	Recovered—21 p.p.—fasting.
10	8	0	170	Abundant	mod.	4-17-22	7.8	After 6 convulsions.
	"	"	–	"	"	4-17-22	7.8	Improved—no more convulsions—5 hrs. later.
	p.p.	–	–	–	0	4-22-22	9.3	Improved—5 days later.
11	7	0	155	0	0	4-10-23	9.4	Normal pregnancy—no symptoms.
	8	"	135	40 G. per l.	marked	5- 3-23	8.6	Eclampsia—after 3 convulsions.
	"	"	140	"	"	5- 3-23	8.4	Eclampsia—after 10 convulsions—at venesection.
	"	"	160	6.5 G. per l.	less	5- 7-23	8.1	Eclampsia—no conv.—undelivered—no sympt.
	"	"	165	10 G. per l.	"	5- 9-23	7.3	Eclampsia—no conv.—undelivered—no sympt.
	"	"	150	10 G. per l.	ascites	5-11-23	8.0	Eclampsia—no conv.—delivered 5 days later.

CHRONIC NEPHRITIS WITHOUT CONVULSIONS

12	9	0	210	+++	slight	1- 4-22	9.6	
13	6	2	–	+	mod.	1-26-22	9.6	Quite toxic.
	7	"	–	+	slight	2-16-22	9.6	Much improved.
14	?	0	160	1 G. per l.	0	2-25-22	8.3	Not severe.
"	"	"	–	–	0	3- 2-22	9.4	Much improved.
15	7	2	220	8 G. per l.	marked	4-26-22	7.2	Ascites. Epigastric pain.
16	4	2	170	trace	0	5- 4-22	12.2	Headache.
17	10	2	160	+	marked	1-14-23	8.7	At term—headache—no other sympt.
"	"	"	175	0.5 G. per l.	"	1-15-23	8.4	At delivery—no change in symptoms.
"	p.p.	"	110	+	0	1-31-23	9.6	16 days later—no symptoms—well.

CHRONIC NEPHRITIS WITH CONVULSIONS

18	10	6	165	+	slight	2-11-22	8.2	After 4 convulsions.
	"	"	150	+	0	2-14-22	8.1	No more convulsions. Child probably dead.
	p.p.	"	–	+	0	2-21-22	9.9	Clinically recovered.
19	p.p.	2	155	1 G. per l.	marked	3-29-22	8.8	4 days after delivery and convulsions.

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blood calcium during pregnancy and believed that their results confirmed the decalcifying influence of gestation, were yet unable to detect any relation between the occurrence of eclamptic convulsions and the calcium content of the blood in the two cases studied.

Halverson and Bergeim[34] noted subnormal serum calcium values in three cases of eclampsia (8.47, 8.5 and 8.66 mg. Ca per 100 c.c.), but in a single case of pernicious vomiting obtained a normal reading of 10 mg. Similarly low calcium figures were noted in many cases of uremia, even though the urinary output of calcium was distinctly below normal, and an increase was detected when clinical improvement of the condition occurred.

Macallum[35] analyzed the plasma of three eclamptics and found "the calcium in two cases above normal and in one case normal; the magnesium in two cases high and in one normal."

Experimental

We have done 36 serum calcium determinations upon 19 patients suffering with various forms of late toxemia (Table IV), and 8 magnesium determinations upon 2 such patients (Table V).

In general, the calcium results were not different from those obtained in uncomplicated gestation, although 3 specimens showed a lower concentration of calcium than any of our normal samples, (Case No. 2, Case No. 11, Spec. 5, and Case No. 15). The first of these patients had no demonstrable edema, whereas the other two had pronounced swelling and ascites. Another unusual case was No. 16, an early pregnancy complicated by a severe grade of chronic nephritis, in which an extremely high value (12.2 mg. per 100 c.c.) was found.

Whenever samples were examined some days after delivery and full recovery, the serum calcium was found to have returned to the normal level, thus following the normal puerperal variations. Case No. 11, was one of interrupted eclampsia, in which labor was induced thirteen days after the convulsions had ceased, because of continued albuminuria and hypertension. Here the symptomatic improvement during the first several days was associated with a gradually diminishing serum calcium in spite of calcium therapy, a fact which argues quite decisively against any direct relationship between the convulsive manifestations and a diminution of the blood calcium. Similar conclusions were reached by Kehrer,[14, 20] and Mazzocco and Moron.[21]

It would seem that the lowered serum calcium in the late pregnancy toxemias is susceptible to the explanation advanced in discussing normal gestation—blood dilution. There is certainly a tendency for the values to be higher when there is no edema and lower when the swelling is marked, although no absolute correlation can be made out.

Our observations upon serum and plasma protein indicate that a greater degree of hydroplasmia is to be expected in toxic women,

TABLE V

SERUM MAGNESIUM IN TOXEMIC PREGNANCY

CHRONIC NEPHRITIS WITHOUT CONVULSIONS

CASE NO.	MONTH OF PREGNANCY	PARA	SYSTOLIC BLOOD PRESSURE	ALBUMINURIA	EDEMA	DATE	SERUM MG	REMARKS
			mm.				mg. per 100 c.c.	
17	10	2	160	++	marked	1-14-23	2.3	At term—headaches.
	"	"	175	0.5 G. per l.	"	1-15-23	2.4	At delivery—no change in sympt.
	p.p.	"	110	+	none	1-31-23	2.8	16 days later—no symptoms—well.
				ECLAMPSIA				
11	8	0	135	40.0 G. per l.	marked	5- 3-23	2.8	After 3 convulsions.
	"	"	140	40.0 G. per l.	"	5- 3-23	3.1	After 10 convulsions—at venesection.
	"	"	160	6.5 G. per l.	less	5- 7-23	2.9	No convulsions for 4 days—no symptoms.
	"	"	165	10.0 G. per l.	"	5- 9-23	2.9	Still undelivered—no symptoms.
	"	"	150	10.0 G. per l.	ascites	5-11-23	2.9	Ascites developed—delivered 5-16-23.

although exceptions are frequently noted. This experience is in accord with Zangemeister[10] and is substantiated by the observations of Stander and Tyler[30] upon the water content of the blood. The latter authors found that in eclampsia or chronic nephritis complicating pregnancy the percentage of water in the whole blood or plasma may be greatly increased, the increase being roughly proportional to the extent of the general edema.

In two patients calcium lactate and calcium chloride were given during the course of treatment and in one instance magnesium sulphate was administered by rectum. No definite change in the patients' condition could be detected, and no real increase in the serum concentration of these substances was demonstrable, although at times it was felt that there was a tendency for the blood pressure to become somewhat lower following such therapeutic procedures.

The few serum magnesium determinations upon toxemic individuals quite consistently gave relatively high results (2.3 – 3.1 mg. per 100 c.c.), with no definite variations incident to the symptoms. Certainly there is in these two cases no diminution of the magnesium, and it may be that further work will confirm our findings and those of Macallum,[35] showing values slightly above normal.

CONCLUSIONS

1. Analyses on 105 normal pregnant women showed a definite and consistent tendency toward a lowering of serum calcium during pregnancy. This tendency was more marked toward the end of the period of gestation; 50 per cent of the women examined in the last 5 months of pregnancy showed serum calcium below the lowest values observed in normal nonpregnant women (9.0 mg. per 100 c.c.), while the same degree of diminution was found in only 22 per cent of those examined in the first half of pregnancy. No values below the normal range were noted in the first 8 weeks of gestation.

2. The average values for serum calcium in normal pregnant women were distinctly lower than the average for normal nonpregnant women (9.7 mg. per cent). They indicate a progressive diminution of serum calcium during the first half of pregnancy and a relative constancy of these values (8.8 – 9.0 mg. per cent) during the latter half of this period and at the time of labor.

3. The decreased concentration of calcium in the serum during pregnancy is probably not related to any calcium deficiency in that period but may be most logically accounted for at present upon the basis of the coincident occurrence of a dilution of the plasma (hydroplasmia), for which there is considerable evidence.

4. The values for serum calcium obtained in cases of the toxemias of pregnancy are quite comparable to those found in normal women

at the same period of gestation. There is no evidence that low serum calcium bears a casual relation to the convulsive manifestations of eclampsia.

5. The serum magnesium in normal nonpregnant women was remarkably constant, 11 out of 16 determinations falling between 2.0 and 2.4 mg. per 100 c.c. (average 2.3 mg. per cent).

6. The magnesium content of the serum showed a tendency to be lower toward the end of pregnancy, and especially at the time of labor, similar to that shown by the serum calcium and probably attributable to the same cause.

7. A few determinations of serum magnesium in cases of toxemia gave values within the upper limits of the normal range.

8. Both the calcium and magnesium content of the serum return to normal shortly after delivery, although determinations made in the first two days postpartum gave values as low, if not lower, than those observed at labor.

9. The serum of lactating women showed no significant variations in calcium and magnesium content from that of normal nonpregnant women.

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AN OUTLINE OF POSTPARTUM CARE*

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THE influence of custom and tradition is shown in obstetrics more than in any other branch of medicine. This is particularly true of the puerperium. Antiquated methods of treatment are still used in an empiric way by many practitioners and it is a fact that practically no two institutions, hardly two individuals, treat postpartum cases alike. In a large majority of cases delivered outside of hospitals these patients may be said not to be treated at all.

The prenatal period approaches a gradual and natural climax, viz., the birth of the baby. Following this event the patient is many times simply placed in charge of the nurse and in the absence of fever or other alarming manifestations, she is allowed to manage the case.

In these days when so much attention has been drawn to the prenatal period and its treatment, it suggested itself to me that it might be well to discuss a few points in the treatment of the postnatal patient. It seems rational to believe that if we are given a normal patient, a normal delivery, and a normal baby, that here at least is one condition in the realm of practical medicine the treatment of which could be more or less standardized. There must be one best outline of treatment for puerperal patients. With this idea in mind I propose to take up in a brief way a few points in the treatment of the normal puerperal patient and to discuss what I think constitutes rational and competent treatment during this period.

What is meant by the puerperal period? It may be stated that this period constitutes the time from the completion of labor until the organs of reproduction return to their normal state, and that this usually takes from six to ten weeks. This return to normal, or involution is most rapid during the first ten or twelve days and while we regard this as a physiologic process it may easily and rapidly become a seriously pathologic one.

The outline of our treatment of this period may be conveniently grouped as follows, 1. To enforce an adequate period of rest.

2. To preserve asepsis in the birth canal.
3. To correctly manage the function of lactation.

Rest.—It is agreed that if there is one person on earth who has earned a good rest it is the parturient woman. An initial period of rest is of very great importance and something which I always insist shall be instituted immediately after delivery. The room should be darkened, the baby and all persons except the nurse excluded and the patient made as comfortable as possible, flat on her back with the head kept

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low. If after a short time a natural restful period is not obtained, I have no hesitancy in using codeine or even morphine in order to secure it. After a long and exhausting labor the patient may oftentimes be too excited to relax into a restful state and an efficient sedative or hynotic at this time is invaluable.

Diet.—Why should not the puerperal patient have a reasonable diet from the start? What is rational about placing the normal postpartum patient on liquids for one or two days and soft diet two or three more before giving them something real to eat? For the past few years in both ward and private patients it has been my custom to give a general diet from the first and I have never seen any untoward results. There is little danger of overeating or digestive disturbances if well cooked food is served to the patient in an appetizing manner. For instance, the diet during the first twenty-four hours may include eggs, toast, ice-cream, bread and butter, cooked cereal, simple salad, baked potato, stewed fruit, rice, and of course soups, water, milk, tea, coffee, cocoa, etc. On the following day a regular diet may be instituted. With sensible reservations the patient may eat anything that she wishes to. It may be furthermore stated that successful lactation is insured by a liberal diet from the first. Our dairy friends who know so much more about the subject of lactation than we do, and from whom we may learn much, agree that a full diet especially rich in protein has an extremely favorable influence in maintaining a rich milk supply. There is also something to be said about the psychologic effect of giving a patient real food when she is hungry. She is more apt to realize that she is convalescing in a normal manner if she is allowed to eat the food to which she is accustomed when up and around.

Bladder.—Diet and digestion naturally lead to the question of excretion. Let me say a few words here concerning the treatment of the urinary bladder in these cases. There is one lesson which we should learn which may be sloganized thus, "Never catheterize except as a last resort." The great danger of prolonged cystitis resulting from catheterization even under the most aseptic conditions must constantly be borne in mind. The patient should attempt early to urinate. If she is unable to do so there are various expedients which may be tried to influence this function. These include warm stupes over the pubes, the sound of running water, and sterile hot water poured over the vulva. Often times a large hot high enema will bring results when other things have failed. We need have no hesitancy about supporting the patient in the upright position by way of further attempt. An odd trick which some of my nurses sometimes successfully employ is that of placing the patient on a bedpan into which has been poured a small quantity of aqua ammonia. Mention should be made of the

hypodermic use of pituitrin .5 to 1 c.c. repeated once or twice at half hour intervals. In patients with marked abdominal relaxation a binder which fits snugly over the pubes often assists by giving the patient "something to push with."

How long may we allow these patients to wait before resorting to catheterization? It is perfectly safe to wait until the bladder forms a definite tumor over the symphysis and in any case 18 or 20 hours may elapse before catheterization is thought of. There is one psychologic point which I have observed in these patients that have to be catheterized, and that is occasionally they become profoundly depressed and unhappy because of their inability to urinate. I mention this so that when it occurs every form of encouragement and assurance may be given them by the physician and nurse.

Care of the Bowels.—The routine use of castor oil and other drastic purgatives following childbirth is as unnecessary as it is unpleasant. Since McPherson[1] made his report in 1917 I have avoided the old regimé of purgation and have adopted the following plan. In normal cases 24 or 36 hours after labor if there has been no spontaneous movement a low soapsuds enema is given. This is repeated daily if necessary. If the bowels seem to act in a sluggish manner a mild vegetable cathartic or mineral oil is given at bedtime.

Let me summarize McPherson's interesting results with regard to this method of treating the bowels postpartum. His experiments were carried out in 1917 at the Lying-In Hospital in New York City and were briefly as follows: In 322 cases in which ordinary catharsis was used 28 had fever at some time during the puerperium. In the same number (322) of cases in which no catharsis was used only three had fever at some time during this period and one of these had a mammary abscess. Allow me to quote the author's conclusions, "When we consider the lessened danger of infection caused by the spreading about the mother's soft parts, of loose diarrheic movements, when we consider the enormous amount of labor saved for the nurses as well as the comfort of the patient, and when we consider the figures quoted in this series, it gives us some intimation of the necessity for active thought on the part of the obstetrician for every move that he makes, every drug that he prescribes, and for less habit and more individualization. The writer is far from believing that there is never any necessity for administering a cathartic in the puerperium; quite the contrary; but what he wishes to emphasize is the danger and uselessness of routine drugging, and the assigning of certain effects to conditions which have not been shown to be the cause of the symptoms exhibited."

It has been shown furthermore by Kettner[2] that milk production is often greatly influenced by catharsis and he states that "a dose of castor oil or any other purge may turn the scale against the normal

development of lactation, especially if the child does not take hold well."

The Birth Canal.—As before noted, our efforts in this regard should be simply to preserve or maintain asepsis. It is essential that the discharges shall be absorbed and not allowed to accumulate. For this reason the vulval pads should be composed of aseptic absorbent material. These should be changed whenever soiled, the number in 24 hours varying according to the amount of discharge. For the first two or three days this should be at least every 3 or 4 hours and each time that there has been urination or bowel movements the genitals should be well washed from above downward. This is best accomplished by vulval irrigation followed by sterile cotton wipes. We may or may not use an antiseptic solution. An antiseptic solution is not necessary as has been shown by E. W. Plass[3] in comparative observations made at the Johns Hopkins Hospital. Here equally good results were obtained, even in cases where there had been perineal laceration, by the use of boiled water. Douches are distinctly not indicated at any time during the normal puerperium. It has been repeatedly shown that infection has followed their use.

Care of the Breasts.—The beneficial influence of lactation on uterine contraction and involution must not be forgotten. After the first rest following delivery the breasts and nipples are thoroughly cleansed with soap and water and a simple ointment applied to the nipples on sterile gauze or oiled silk or paper. Either sterile vaseline or lanolin may be used for this purpose. Eight or ten hours after birth the baby should be put on the breast. This early initiation of nursing is very valuable for a number of reasons. The uterus by reflex stimulation expels clots more easily and any tendency to hemorrhage is checked. The milk production is augmented and the colostrum which acts as a natural cathartic prepares the baby's intestinal canal for milk digestion.

It does not seem out of place at this point to speak concerning some very interesting work which has been recently reported concerning the significance of colostrum in newborn calves. Theobald Smith and R. B. Little[4] have demonstrated conclusively the importance of this fluid at least for newborn calves, and I think we may draw an important analogy from their work. Because of the difficulty in keeping alive calves which have not received colostrum, these investigators experimented with two groups of animals. In one group of ten calves which received colostrum, they report three deaths. In a group of twelve calves which did not receive colostrum they report nine deaths. Their conclusions were that the calf which is deprived of colostrum lacks something which permits intestinal bacteria to invade the body and multiply in the various organs, and they further conclude that the

function of colostrum is essentially protection against the miscellaneous bacteria which are harmless later on when the protective functions of the calf have begun to operate.

It is my custom to place the baby on each breast for five minutes every four hours until the secretion of milk begins to take place. At this time three hour intervals are assumed in the daytime and four hour intervals at night. Nothing further is done for the breasts except cleansing before and after each nursing with boric acid solution.

If the breasts are very pendulous a light binder may be applied, never so tight as to interfere with the free access of air. At the beginning of lactation the breasts very often become engorged and painful. As a rule this is largely due to venous congestion and not to milk retention. When this occurs fifteen or twenty minutes' steaming with a large hot compress of boiled water will relieve the pain and congestion almost immediately. Occasionally a single dose of codeine may be given in conjunction. I never use the breast pump for this purpose for this appliance unquestionably stimulates the breast to further activity a short time after it is used. If fissures are treated immediately on their appearance, breast infection will seldom occur. Various astringents such as glycerite of tannin, tincture of benzoin, or witch hazel may be used. If relief is not secured in a very short time the use of the nipple shield should be begun immediately. A glass shield is used similar to that at the Sloane Hospital, in combination with a three hole anticolic nipple. Of course this is boiled before each nursing. If the fissures persist and become deep it may be necessary for a light cauterization with the silver stick and the restriction of nursing on that side for one or more periods. Mention should also be made of the use of the lead nipple shield which I have sometimes used with success.

The Abdominal Binder.—"To bind or not to bind that is the question." The proper treatment of the relaxed abdominal wall following childbirth demands our attention not only to prevent a pendulous abdomen but also to secure comfort for the patient. While I do not advocate the routine use of the abdominal binder I do not think it can be entirely dispensed with. This is particularly true in multiparæ where there is sagging and relaxation of the abdominal wall. In these cases no amount of massage or abdominal exercise is going to have appreciable effect at least during that part of the puerperium which is spent in bed. These patients need abdominal wall support not only for comfort but to help them during defecation and micturition, to give them something to push with. The binder should be well fitted and not too tight. I do not believe all the dire results that have been attributed by numerous writers to the use of the abdominal binder, such as compression of the uterine and ovarian veins, retardation of

involution, constipation, subsequent prolapse. After the patient is up and about it seems to me time enough to begin the kind of abdominal exercises which will prove really beneficial in restoring muscular tone to the abdominal wall. At this time also I think that a properly fitting corset is essential, particularly in cases where a corset has been worn previous to delivery.

When Should the Patient Get out of Bed?—There have been more various opinions and more discussion about the length of time the patient should remain in bed following childbirth than any other question concerning the puerperium. Writers have advocated everything from keeping patients in bed three weeks to getting them out of bed 48 hours after delivery. I think very little analogy may be drawn from animals or primitive races in this regard. With regard to the former an entirely different mechanical problem presents itself. In human beings the floor of the pelvis in the erect posture is subjected to a downward pressure which does not obtain in animals in the quadrupedal position. In the latter, a careful study of the differences in musculature, carriage, habits, and athletic ability between these and their more civilized sisters will soon demonstrate why the latter should not go about immediately after delivery.

I think we should first consider posture in bed. Here one of our first problems is free drainage and if the patient is kept lying on her back the entire time free drainage is impossible. In order to insure this in normal cases on the fourth and occasionally on the third day I begin propping them up in bed. The general rule that I follow is as follows: The first few hours after labor the patient is kept flat on her back without pillows. After 24 hours she may turn on her side for a short time. After the third day she is encouraged to lie on one side and then the other and for a while each day on her abdomen. On the third or fourth day she is propped up for a short time one notch on the Gatch Bed. This is increased daily so that by the seventh day the patient is sitting up in bed practically ad libitum. If on the tenth day the fundus has reached the level of the symphysis she is allowed out of bed in a chair a few minutes. This is increased daily as the patient's strength permits and the thirteenth or fourteenth day a few steps are taken.

Three weeks at the hospital is not only not excessive but quite ideal and when patients insist on going home before this time careful instructions are given so that the hospital regime shall be continued at home. Patients are not allowed to climb stairs under three and one-half weeks and are allowed to ride in the open air after the fourth week.

As soon after this time as the patient is able to come to the office I make the final examination. At this time a thorough pelvic and gen-

eral examination is made and the case referred to the family physician if no further treatment is necessary.

In this somewhat discursive paper I have tried to emphasize the main points in the care of the normal puerperal patient. It is only the careful study of a definite postpartum regime that the physician will do his whole duty to his patient. If he will take the time to explain the reasons for exacting care at this time he will find that every intelligent patient will willingly cooperate.

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59 COLLEGE STREET.

THE FEEDING OF PLACENTAL EXTRACT TO MOTHERS; ITS EFFECT ON BREAST-FED INFANTS.*

BY SAMUEL F. ABRAMS, B.S., M.D., ST. LOUIS, MO.

THE poor success which a large percentage of my recently delivered patients had in nursing their babies, induced me to look into the question of stimulation of the flow of breast milk. The placenta has received a great deal of experimental attention, since it develops concomitantly with the marked growth of the breasts. Whether or not the placenta is a secretory organ is a subject much discussed in the literature, but more recently the majority of investigators seem to agree that the placenta elaborates an internal secretion.

The placenta was first described as an internal secretory gland in 1884 by Johannes Miller, and since then a great deal of literature, both for and against, has accumulated on the subject.

Bandler, in the New York Medical Journal, November, 1920, says: "Although not making a plea for the use of placental extract, it must be considered as an internal secretion, the same as thyroid, ovary, and other glands; and as to its use being empirical, so is that of all the other glands." He theorizes at some length to show the balance of the various glands and their effect on menstruation and pregnancy; all of which may be summed up as follows: We are dealing with a secretion produced by the outer shell of the ovum, viz., syncytial cells and chorionic epithelium, which is the early placenta. From the beginning of pregnancy this placental tissue is thrown into the blood stream, producing a secretory action. The corpus luteum prevents menstruation, holding the decidua for the fertilized ovum. The corpus luteum must be stimulated to added growth during pregnancy to prevent menstrua-

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tion and miscarriage; and the stimulus is supplied by the secretion from the early placenta, which changes the false to the true corpus luteum. The posterior pituitary gland is the antagonist of the corpus luteum; when stimulated by the growth of the corpus luteum, it causes the onset of menstruation. The placental secretion aided by the corpus luteum is antagonistic to the overaction of the posterior pituitary gland, and prevents the arousing of the posterior pituitary, thereby inhibiting menstruation and miscarriage. There is a balance of the endocrines in the menstrual cycle, the ovary and posterior pituitary being balanced by the thyroid and corpus luteum. During pregnancy these are held in check by the placenta. Thus Bandler tries to prove a secretory action of the placenta.

Fellner, Halban, and Cohn, obtained results showing that the placental extract plays a rôle as a stimulus to mammary growth in the virgin; while Biedl, Königstein, Basch, Frank, and Starling, in experiments seem to disprove this idea.

Hammett, in a series of pregnant women in the Boston Lying-In Hospital, could detect no difference in size of the breasts when feeding placental extract, and says: "From the evidence at hand it is reasonably certain that the placenta has little, if any, direct influence upon the mammary hyperplasia of pregnancy, the source of the stimulus lying in part in the fetus."

Mammary hyperplasia represents the preparation of the glands for the function of secreting milk; and the source of the stimulus initiating and maintaining lactation is the problem to be chiefly discussed here.

Studies of Goetz, Ribbert, and Hister, show that nerve stimulation is unimportant for milk secretion. Halban, in a series of experiments, eliminated ovaries, uterus, and fetus as the immediate cause, and concluded that the placenta, during pregnancy, gives off a substance acting as a check to milk secretion, and, therefore, removal of placenta at term allows the secretory function to be taken up. On the other hand, Nicklos claims to have obtained increased milk secretion with placental extract, and concludes that there is an overflow of placental secretion into the blood stream during labor. This causes milk secretion. This explanation, however, does not take into consideration the interval between labor and onset of lactation, nor the question of the continuation of milk secretion.

Van Hoosen and Cornell conclude that placental extract acts as a galactagogue, basing their conclusion on the increased weight of the infants.

In a series of cases published in the Woman's Medical Journal, Van Hoosen gives some very striking results, and calls attention to the fact that many animals eat their placenta. She also emphasizes an-

other difference between the human and animals; namely, that the young animal immediately attacks the breasts, thereby directly stimulating them. She states: "The first report of our results came in the form of a complaint from the nurses. They requested that the dose be cut down because the patients had so much milk that it was a burden to keep the breasts empty. The head nurse on the floor reported that on the morning this complaint was made, there had been removed from a patient 16 ounces of milk after the infant had taken all it would. Three other patients had each 6 ounces removed after nursing; a fifth had 8 ounces, and a sixth, 4 ounces. A total of nearly three pints of breast milk in one morning from six patients, without depriving the baby."

In this paper 25 cases are tabulated, all the patients having received scopalamin—morphine analgesia, and placental extract. The results were as follows: One-fourth of the infants gained during the first week, and one-half of the infants maintained or increased their birth weight at the end of the second week. Only two infants lost in weight, both first and second week. That this effect cannot be ascribed to the twilight analgesia, she proved by running parallel series of cases, both without placental administration, but giving one series scopolamin. She found that only one infant gained the first week, while four infants lost both first and second week.

In a series of 300 cases of McNiel, and McNiel and Hammett, the nursing women were fed 30 grains of desiccated placenta per day, without an increase in milk secretion. In another series by the same men, the chemical composition of the milk was tested after the feeding of the desiccated gland. They found a slight decrease in the percentage of fat, a slight increase in protein, and a rather marked increase in lactose.

Although, there was no actual increase in milk secretion, a series of cases reported by Cornell, showed that 87 per cent of the babies, whose mothers received desiccated placenta, began their gain on the fourth and fifth day of life, against 69 per cent of those not receiving the placental extract; 44 per cent of the former regained their birth weight before leaving the hospital against 24 per cent of the latter. He found the age of the patient played no part in the above series.

Hammett concluded that placenta does not act as a galactagogue, but as a stimulus to the infant's growth, and in a large series of cases showed that there was a smaller postnatal decline, a quicker recovery from initial loss, and an increase of 60 per cent of the rate of growth by the thirteenth day.

To ascertain the effect of the administration of placental extract on the maternal metabolism, the purin metabolism of a series of cases

was determined by Harding and Young. They found a very marked increase in purin metabolism, too marked to be ascribed to the purin contents of the ingested placenta. Whether or not placental tissue actually causes an increase in milk secretion, the changed milk composition and increased purin metabolism certainly speak for some hormone action.

I believe the most important and clear cut results of any investigator were obtained by O. Frankl. He showed that the placenta is an internal secretory organ and by its action inhibits the formation of milk. He grafted placental tissue into mice just before the birth of their young, and found that wherever there was a "take" of the graft, the young animals died of inanition due to a persistence of the colostrum stage.

The proper dosage with which maximum results could be obtained was also a matter of discussion. Cornell said that he obtained his best results with a dose of 5 grains of desiccated placenta three times a day. Van Hoosen began with 50 grains per day in divided doses. Her patients complained of headache, so the dosage was cut to 30 grains per day.

In the series of cases which I wish to report, a large number were given 60 to 80 grains of desiccated placenta per day by mouth without complaint. The average dose of desiccated placenta for most patients in this series was 10 grains three times a day. About 10 patients, not included in this series, received "Placental Hormone," an experimental product of Parke Davis & Company, but this was discontinued because the patients objected to the discomfort of the hypodermic injection. These cases are not included because there were too few to allow conclusions.

My series consists of 76 cases from the service of the St. Louis City Hospital. Any markedly abnormal cases, such as puerperal infections with high temperatures, or mothers with babies so premature and small that they could not nurse, were excluded from the experiment.

On account of one's inability to forecast the supply of milk, it was very difficult to interpret results. It seemed best to compare a fairly large group of cases with another group which did not receive placental tissue, but whose care and food otherwise were the same. This was done by checking against a group of normal cases of another obstetrical unit of the hospital which were delivered about the same time.

All cases were charted, one curve showing the weight of the baby, another its temperature, and a third the mother's temperature. The amount and kind of all medication or abnormality was noted on the chart.

Since the large babies as a rule nurse better than the smaller ones,

and as the better stimulation makes it likely that the mothers of these babies would have a greater supply of milk, the average weight of the babies in both groups was compiled to see that there was no marked weight advantage of one group over the other.

The average weight in those receiving medication was 3278 grams, and in the other group was 3339 grams, a difference of 61 grams.

The average initial loss of weight in group I was 243 grams, and in group II, without placental medication was 314 grams. Thus with the average weight of the babies in both groups practically the same, the average initial loss was greater in group II.

In checking the amount of gain over birthweight in group I, I was rather surprised to find that although more babies regained their birthweight by the tenth day, the amount of gain was less than in those of group II who did not receive placenta. The average gain over birthweight in the first group was 57 grams, while in the second group it was 142 grams. Although the amount of gain in the second group was greater, there was a marked difference in the number who regained their birthweight. By the tenth day 49.7 per cent of the first group had regained their weight, while only 30 per cent regained it in the second group.

It has been claimed by some that the mother's milk "comes in" sooner when placenta is fed before delivery. I was unable to substantiate this claim, as feeding for a week or 10 days before the birth of the baby did not cause milk to appear before the third day.

There is, however, some difference in the time at which the babies begin to gain weight. Taking the fourth and fifth days together, 86 per cent of cases of the first group gained weight, while only 68 per cent of cases of the second group gained. Sixty and three tenths per cent began to gain weight on the fourth day of life, when the mothers received placenta, while only 30 per cent gained on the fourth day, when no placenta was fed.

Although the results are not striking, there is enough difference to believe that the placental extract has had some action on the growth of the babies, even if not on the increase in breast milk. Certainly, there is a definite difference in the averages of the two groups of cases.

In order to check up the above series and to see if the results may not have been accidental, Dr. Storrs kindly consented to a similar investigation at the St. Louis Maternity Hospital. This series consisted of only 15 cases, but the general results were about the same as in those reported above, although the increase over birthweight was markedly less in all of these cases, as compared with both City Hospital groups.

In group I, (those to whom placental extract was fed), 33 per cent

gained on the fourth day, 66 per cent on the fifth day, and none later than the fifth day. Four cases, or 26 per cent, regained their birthweight by the tenth day.

Group II (no placental feeding), showed 26 per cent gain on the fourth day, 60 per cent on the fifth, and 13 per cent on the sixth day. Taking the fourth and fifth days in this series, 100 per cent gained in group I, and only 86 per cent in group II.

The average weight of the infants in the two groups of the St. Louis Maternity Hospital series was 3475 and 3438 grams, respectively.

The difference in the average initial loss of weight was very small, but still in favor of group I. It was 247 grams for group I, and 261 grams for group II.

The age of the City Hospital group of patients was between 16 and 30 years, and about the same number of primiparae as multiparae, some of the latter giving a history of no milk with their previous pregnancy, and a good supply this time, and vice versa.

To draw conclusions from a series of this kind, many things should be considered, and I believe among the most important is the psychology of the patient. Those at the City Hospital are of the stolid type and desire to nurse their babies, while most of those in the wards of the St. Louis Maternity Hospital, are highly neurotic, besides which, they are very choicy of their food and eat less.

The size of the breasts has some influence, but does not determine whether or not there will be a sufficient milk supply, as many of the small breasts secreted better than the larger. Some breasts were so small and flat that when filled to capacity they had only about an ounce of milk.

The two series of observations reported here closely resemble those of Cornell and Van Hoosen in many ways. I feel that although all these investigations show that placental medication does influence either milk secretion, or the immediate growth of the infant, neither a real galactagogue, nor the main stimulus of milk secretion has been discovered in dried placental tissue. The results have not been of a sufficiently clear cut character. I believe that if the placental secretion really stimulates the secretion of milk, the feeding of large amounts should produce the effects which could be seen grossly and without question in most cases, especially in those which already have a large amount of milk and large breasts.

If Frankl, Fellner, Halban, and Cohen, are correct in their theory, that the hyperplasia of the breast is induced by the placenta and that something else initiates and maintains the milk supply, might it not be well to feed flat-breasted prenatal patients with desiccated placenta? If Frankl's experimental results are true in the human, then the apparently somewhat favorable results in the above series demand some explanation. This probably can be done in the fol-

lowing manner: With the placenta antagonistic to the organ causing milk secretion, its removal allows this organ to functionate. Small amounts of placenta given at that time, not enough to definitely inhibit the function of this organ, might cause some added hyperemia in the glandular tissue, and thus cause the change in milk composition and a slight increase in milk secretion.

Up to the present time, however, by far the best stimulus to milk secretion has been found to be a large hungry baby and a cooperating mother.

METROPOLITAN BUILDING.

PAINLESS CHILDBIRTH BY SYNERGISTIC METHODS*

(A Preliminary Report)

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MYTHOLOGICAL, profane and sacred literature abound in incident, fact and fancy, showing that from the dawn of history man has sought to assuage grief and pain by some means of dulling consciousness.

Both the Bible and the Talmud contain references to the ancient practice of inducing sleep by artificial means. In these attempts many methods and diverse agents have been employed. The inhalation of fumes from various substances, weird incantations, the external and internal application of drugs and many strange concoctions, pressure upon important nerves and blood vessels, mesmerism, hypnotism, etc., have all played their part in the evolution of anesthesia. But modern anesthesia was not known to the world until after the successful public demonstration of ether by Morton in 1846.[1]

One of the first uses to which it was put was to modify the pains of childbirth. Sir J. Y. Simpson[2] of Edinburgh, Scotland, was the first to use ether in obstetrical cases, but not entirely satisfied, sought a substitute and to him we are indebted for the second most universally used agent for general anesthesia—chloroform. Common sense prevailed over the opposition of clergy and laity of his day, "to avoid one part of the primeval curse on women," and chloroform and ether are now used almost universally for special cases and in certain stages of labor. However, where expense is not considered, nitrous oxide and oxygen have supplanted the use of these agents.

In 1880, Klikowitch[3] of St. Petersburg recommended an 80 per cent mixture of oxygen with nitrous oxide for obstetrical purposes and this is about the admixture that is used today for analgesic purposes. Guedel of Indianapolis was one of the first to endorse strongly this combination in normal labor in the United States (1911), and it is now quite generally employed.

Spinal analgesia has been used with success—as have also local analgesics to the

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cervix, the perineum and the rectum, but these methods have but few followers today.

Schneiderlin, in 1899, recommended the use of morphin and scopolamin for surgical anesthesia, but Steinbüchel first recommended its use in labor in 1902. Krönig and Gauss of Freiburg are responsible for its introduction into the United States, reporting 3,000 cases in a paper read in Chicago in 1913. Krönig and Gauss use narcophin, a proprietary narcotin—morphin meconate. There is a good reason for using this combination and those who do not, have no right to condemn or criticise "twilight sleep". Sollman states, (P. 260) "Narcotin has a considerable potentiating action on morphin. The hypodermic injection 5 mg. of morphin produced no measurable analgesic effect with 10 mg. the analgesia was marked in two subjects; the third responded by hyperesthesia. With narcotin 8 mg. was ineffective, 30 to 40 mg. produced first some hyperexcitability then slight analgesia. With a mixture of equal parts of morphin and narcotin containing 3⅛ mg. the analgesia was almost as great as with 10 mg. of morphin alone. This combination was also effective on the subject who resisted morphin." (P. 267) "Morphin probably tends to delay the progress of labor by its psychic sedative action, largely by preventing the reinforcement of labor pains by the contraction of the abdominal muscles."

This system has been in turn, praised and condemned; today it is practically abandoned except by Krönig and Gauss and a few specially equipped sanitaria.

Sollman further states, "the use of scopolamin-morphin anesthesia is justified (if at all) only in specially equipped institutions and not in private practice, or even in ordinary hospitals." It is applicable only in about 30 per cent of labor cases.

All of the above agents and methods require either a specialized technic, unusual equipment or trained personnel. Numerous inquiries from many sources indicate that none of these methods are satisfactory to the majority of physicians.

The development of painless labor by synergistic methods was undertaken from a purely scientific and altruistic viewpoint. It was decided that the method should be so simple that it could be used either in the home or hospital, and by any physician, in an entirely empirical manner. The ideal sought was a state of relaxation and analgesia with consciousness, but little if at all impaired, so that full cooperation might be had at all times. The methods by which this condition was to be obtained were outlined to the chief and attending obstetricians of the Lying-in Hospital, Asa B. Davis and George W. Kosmak, and were as follows:

1st. To start with a minimum dose at which time no definite results were expected and to gradually increase the dose until definite results were obtained.

2nd. To stop at any time in the development of the technic, if either mother or child appeared to be in danger.

3rd. To publish the results whether favorable or unfavorable.

With this understanding, work was commenced on the 10th of February, 1923. The first 4 cases showed practically 75 per cent failure. Today the first chapter of painless labor by synergistic methods is completed when we can secure over 75 per cent success. By "success" is meant the amelioration of pain to such an extent that patients

state that they were "helped,"—only a few of the 75 per cent had a comparatively painless labor. By "failure" is meant whenever the patient obtained no relief from the method. At no time was the mother's condition imperiled in the slightest degree. Only in one instance did the infant seem to be affected. Whether or not this was from the medication cannot be stated positively, but it seemingly was. In the first series of drugs used, there was not only failure but delay. This delay was in a measure corrected by placing 10 grains of quinine hydrochloride in the mixture. Sollmann states that "satisfactory results have been reported from its use in all stages of labor. This drug stimulates the contractions and increases the tone of the uterus."[4] Since this addition, in only one or two cases has there been any dedelay, and in these exceptional cases, exhausted nature was more powerful than the drug.

There also seemed at one time to be an increase in the nausea and vomiting. This was corrected in a very great measure by eliminating urethan and paraldehyde, thus also making the final mixture simpler. The other drugs considered fundamental to the scheme are magnesium sulphate, urea, ether and morphin. Two to four drams of the magnesium salts is the maximum dose used in our investigations of painless labor, although the writer has used two ounces of the salts *per rectum* in other cases without deleterious effect. The magnesium sulphate must be chemically pure (the usual commercial product may cost 10 cents per pound—the chemically pure $1.00 per pound).

Weston and Howard[5] have injected 2 c.c. or more of a 50 per cent solution of magnesium sulphate subcutaneously or intramuscularly more than a thousand times with no local pain or sloughing. They state the sedative action occurs in fifteen to thirty minutes and lasts from five to seven hours, and is found to be a very good substitute for morphin and hyoscin. In a few instances the patient became quiet but did not sleep. In 82.7 per cent it was effective. In 6 per cent the dose was repeated before sedation occurred. In 11 per cent no effect was noted even after three or more doses.

Rector[6] has used magnesium sulphate colonically in doses of two drams in over 300 cases. The Presbyterian Hospital used the salts over 200 times by hypodermoclysis in four dram doses with good results. In both instances other drugs or agents were added to complete the analgesia or anesthesia.

Magnesium sulphate used in two to four dram doses by rectum leaves a margin of safety amply sufficient to satisfy the most critical and is, next to urea, probably the safest drug in the combination. In the final series this drug is given hypodermatically.

In a personal communication, Alma J. Neill, Professor of Physiology, University of Oklahoma, states: "I have found that the rate of diffusion of the magnesium sulphate with urea was rather constant; that is, the range was between 30.9 and 32.1 per cent with the exception of a 6 per cent solution of the magnesium sulphate and a 1 per cent solution of the urea which diffused very much faster, it hav-

ing diffused 48.1 per cent in the same time. The whole range of percentages was used both with the magnesium sulphate and the urea. Each time the 6 per cent solution of the magnesium sulphate and the 1 per cent solution of the urea diffused practically 50 per cent faster than any other combination or any other substance which I tried."

Hewlatt[7] has given 25 grams of urea every hour until 12 M. (100 grams in all) with no untoward results. Cushney[8] states: "It is rapidly absorbed from the intestine and is practically devoid of action in the tissues even in the larger doses." The amount used, 1 per cent of a 4 ounce mixture, is therefore negligible as far as danger is concerned. It is used with the idea that it increases the absorbability of the magnesium sulphate. The exact relation between "diffusion" and "absorbability" is not definitely known, nor can we state at this time how much the efficiency of the method is due to the urea. Urea is not used in the last series.

"Ether[9] is the anesthetic of choice if the patient is suffering from any form of toxemia or requires stimulation or is suffering from shock." The amount used in our mixture, two to four drams to a maximum of not over three ounces needs no extended comment. (Four to six ounces of ether is the amount used in colonic anesthesia.) Ten years after its introduction for general surgery, oil ether was first systematically used in 100 obstetrical cases by Thaler and Hübel.[10]

The mixture was 90 grams (3 ounces) of ether and 120 grams (4 ounces) of olive oil. The amount used at one injection was 100 c.c. (3½ ounces) introduced very slowly. In a typical case, a few minutes after injection, the eyelids close and a general relaxation sets in. The condition is suggestive of twilight sleep, but if there is no marked effect after ten minutes, a second injection of 50 c.c. (1¾ ounces) is given. In only one case was the method entirely impractical. With this one exception there was no complaint of pain or irritation of intestine.

In	16	cases	this	injection	(50 c.c.)	was	repeated once.
"	25	"	"	"	"	"	" twice.
"	20	"	"	"	"	"	" three times.
"	15	"	"	"	"	"	" four times.
"	12	"	"	"	"	"	" five times
"	4	"	"	"	"	"	" six times.
"	2	"	"	"	"	"	" seven times.
"	1	"	"	"	"	"	" eight times.

In one case the maximum total amounted to 767 c.c. of which 270 c.c. were lost. No rectal irritation.

In 88 cases the results were satisfactory.

In 4 cases there was absolute failure.

In 80 cases normal or very strong labor contractions.

In 20 cases labor was reduced or retarded. In these cases quinine or pituitary extract was added.

In some cases the labor seemed to be improved by the oil-ether mixture.

In 73 primiparas average duration of birth 20-¾ hours

In 27 multiparas average duration of birth 10-¼ hours

No anomalies of the afterbirth period were observed. No change in fetal heart beat. Usually born pink.

In 84 children condition normal—cried immediately.

In 14 children condition apnoeic—breathed normally in five minutes without resuscitation.

In 2 cases typical asphyxia—1 revived and second—forceps—was not resuscitated.

In the majority of cases during the intervals between labor contractions, the pa-

tients lay as if asleep, during labor contractions slightly restless and groaned occasionally.

The time of rectal instillation:

In 24 cases the os was dilated 2-4 fingers

In 27 cases the os was dilated 3 fingers

In 9 cases the os was dilated 4 fingers

In 3 cases the os was dilated more than 4 fingers.

No morphin or alkaloids were used. In no case was there excitement. Vomiting in five cases. Strong thirst in seven cases. After delivery a deep sleep. Upon awakening do not recall any incidents. Of 99 born alive, 1 child died on fifth day, after third day intestinal inflammation and subsequently pneumonia. Some babies were sleepy during the first one or two days. Ether by inhalation in comparison with this method affects the brain too much.

The disadvantage might be the impossibility of exact dosage on account of loss, but according to the writers, the difference of individual reactions makes exact dosage irrelevant. The method is impractical in private homes.

To revert to the synergistic method again the next drug to consider is alcohol. The ounce of alcohol was used principally as a vehicle for the ether; it also increases the absorbability of the mixture. Alcohol is below ether in analgesic qualities. Three-eighths of a grain of morphin *per rectum* (in our opinion) is the limit for this drug. The hyoscin is used to accentuate the value or to synergize the morphin. The alcohol was later reduced to two drams.

"The exaggeration of the effects of small doses of morphin which results from its combination with scopolamin are of great practical importance. This synergism may be experimentally demonstrated in various species of animals, especially in those species in which scopolamin alone, even when given in large amounts, produces no narcotic effects. The combined administration of small doses of morphin and small doses of scopolamin, which by themselves produce hardly any effects, results essentially in an exaggeration of the effects of morphin." (Burgi, Madelung.) Morphin and the hypnotics of the alcohol group when administered simultaneously also act synergistically, with a resulting exaggeration of each other's pharmacological actions. (Burgi, Fuhner.)"[11]

THE METHOD

It is the exceptional patient who is so unfortunate as not to have received instructions, among other things, to keep the lower bowel clean, especially in the last stages of labor. The rule at the Lying-In Hospital is to examine and give a cleansing enema when admitted to the hospital. The patient is then sent to the floor where delivery takes place. The patient is therefore already prepared for the colonic administration of the drugs used, and this is the method adopted. A six ounce mixture seemed preferable, but was too frequently expelled, therefore a four ounce mixture is the final choice.

Time.—In selecting the time for the rectal instillation, we seemed to follow naturally the Freiburg method which is as follows: "After labor is well on its way, when the pains are four or five minutes apart and lasting thirty or more seconds, the first injection (hypo-

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dermic) is made." It is at this time that the rectal instillation is given whether in a four ounce mixture or in divided dosage of two ounces. The effect is noticeable about the same time as a hypodermic would be and is not as painful. Analgesia is present in from thirty minutes to one hour after the administration, even in those cases in which a delayed action occurs. The time factor is subject to change.

Suitable Cases.—In the development of this method great care is being taken in rejecting cases that would in any way obscure the issue. For instance, if the uterus is dilating evenly and the contractions occur regularly, but with little pain, no medication is given; an even and sometimes painless delivery is then assured; or, when the cervix is fully dilated (four fingers or more) medication is withheld. Again, if the fetal heart sounds are irregular or bad, or malpositions occur, or if there is any question about the condition of the child, medication is withheld. After starting this method, a patient with indistinct fetal heart sounds was admitted to the hospital. Medication was withheld and the child delivered, but died within forty-eight hours. If the method had been tried in this case it would have properly come under suspicion. The cases selected were those not too far advanced and where there was a possibility of helping them. The selection or rejection of these cases necessarily fell upon the House Surgeons of the Lying-In Hospital to whom full credit is herewith given in exercising unusually good judgment.

Results.—The results have varied, but in the majority of cases, the patients have been "helped," the "pains were lessened" and, in a few, a comparatively painless delivery has occurred. Others were not helped in the slightest, while one or two stated that the pains were "intensified." This last statement can only be accounted for by taking into consideration the mentality of the patient who possibly expected very great help and received but little.

Formulae Used.—After using over fifteen different formulae *the procedure was changed entirely as follows*: The magnesium sulphate is now given by hypodermic and the rest of the medication by rectal instillation. Formula number 16 is 2 c.c. of a twenty-five per cent solution of magnesium sulphate (chemically pure) and morphin is added to the first hypodermic when indicated. Hypodermic is repeated once or twice but without the morphin. The rectal instillation used is:

Quin. H. Br.	Grains	10
Alcohol	Drams	4
Ether	Ounces	2½
Olive Oil	Ounces	1

Reliance is placed upon the synergism of ether and magnesium sulphate for the major effect.

Cowan states that, "in nearly all cases the cervix has dilated with

or without the addition of morphin but dilatation has progressed faster with the thick tough cervix when morphin has been given with the magnesium sulphate. In excitable patients and where you expect a long labor dissolve ¼ grain of morphin in the first hypodermic of magnesium sulphate. The ether, dissolved in one-third the amount of olive oil is given by rectum but not to the extent as used by Thaler and Hübel. The ether is not repeated as it is retained in over 98 per cent of the cases. The following is the usual procedure: Hypodermic magnesium sulphate (chemically pure) 2 c.c. of a 25 per cent solution (with or without morphin) is given when the cervix is dilated about two fingers, and repeated when necessary—one to three times. One to two hours later the instillation is given, when the pains are four or five minutes apart and lasting 30 or more seconds, and when the cervix is dilated two and one-half to three and one-half fingers.

This present technic is far superior to anything we have heretofore used, and with the last forty patients has been changed only in minor details.

Normal cases, not too far advanced, are selected as otherwise the issue would be obscured. The usual method is as follows: Place patient in Sims position and insert small catheter filled with olive oil (to exclude air) three or four inches in rectum. A small glass funnel is attached to free end of catheter and is held just above the hips; the mixture is now poured in slowly. Pressure is made during pains with a crumpled towel held in the hand pressed firmly against the perineum to prevent expulsion.

Cowan improvised a technic for parturient women, for which he cannot be given too much credit, and which is vastly superior to the funnel gravity method just described, inasmuch as it takes less time, is less likely to be expelled and by forcing the mixture into the upper portion of the rectum it is more quickly absorbed. It converts an uncertain procedure into a certainty and is as follows:

Cowan's Technic.—Connect catheter with large syringe holding the mixture, instil slowly, and with gentle pressure force the fluid between pains, the whole amount to be instilled between two and three pains. During pains pressure upon the perineum as heretofore.

This procedure should not take over five minutes, but pressure on the perineum should continue for fifteen minutes more. The advantages of this technic are obvious. The descending head tends to occlude the rectum and make instillation by gravity long and difficult, but with a syringe we can force a considerable part of the fluid beyond the head into the upper portion of the rectum. With this method the whole amount of fluid is retained in over 95 per cent of the cases. Relief of pain is noticed in from fifteen to forty-five minutes, the pain-free period lasting from four to five hours.

Results.—The effect of the synergists upon the nervous system is exciting in 3 per cent of the cases, unchanged in 3 per cent and sedative in 94 per cent.

It was thought this excitement was due to the alcohol, which is now reduced from four to two drams. There has not been a sufficient number of cases since to determine the effect of this reduction.

The deliveries are 4 per cent with forceps and 96 per cent normal.

Occipito-posterior positions rotate in about the same proportion with as without the synergists.

Comparatively, postpartum hemorrhage is unchanged.

Labor is increased in time in 4 per cent, and progresses uneventfully in 96 per cent of the cases.

Nausea occurs in 2 per cent, thirst in 4 per cent, vomiting 8 per cent and normal and uneventful in 86 per cent of the cases.

Pain is increased in 1 per cent, unchanged in 3 per cent and modified or painless in 96 per cent of all cases.

Condition of Baby.—Asphyxia (not fatal) 1 per cent, apnoeic 2 per cent and crying 97 per cent.

CASE REPORTS

From service on Fifth Floor (O'Reagan)

With the present technic pain is materially decreased in all cases, and in a few entirely eliminated: and labor is not, as a rule, delayed or prolonged, any more than it would be if the drugs were not used.

From service on the Sixth Floor (Cowan)

July 13, 1923. Mrs. R. W., Multipara. All previous labors normal. When admitted severe pain every two or three minutes and lasting for one minute. Cervix 2 fingers dilated, thick and soft. Ampoule of magnesium sulphate (2 c.c. 25 per cent solution) given at time of instillation. Distinct sedative effect lasting four and one-half hours. Patient stated she felt very little pain but was conscious that contractions were continuing. Second ampoule of magnesium sulphate given but no decided effect. Patient had fairly strong pains for two and one-half hours before delivery. Baby crying when delivered.

Conclusion—Morphin should have been given with first ampoule of magnesium sulphate, but instillation should have been delayed until cervix was dilated to about three fingers.

July 31, 1923. Para 1, Mrs. I. V. Colored. Moderate sized woman who cooperated well when admitted, was having strong pains every two minutes, lasting fifteen seconds. Cervix three fingers dilated, soft, thin. Hypodermic 2 c.c. magnesium sulphate 25 per cent solution and at same time instillation was given, syringe method. Whole amount retained. Distinct sedative effect. Pains practically subsided, but patient felt contractions continuing. Patient dilated fully with the synergists. No nausea, or vomiting. Patient delivered one and one-half hours after instillation. As head was passing over perineum patient had considerable pain for five minutes. Excited. First degree laceration. No chloroform given except for repair. Baby crying when delivered.

Conclusion.—This patient should have had morphin with first hypodermic and also chloroform during last five minutes of delivery.

July 14, 1923. Mrs. L. S. Para 1. Medium size, pains every five minutes, fairly strong and lasting fifteen to thirty seconds. Cervix three and one-half

fingers dilated, soft but not obliterated. Hypodermic and instillation given at same time. Sedative effect lasted three hours and twenty-five minutes when hypodermic was repeated with good synergistic effect. Thirst and vomiting present. Cervix dilated from three and one-half fingers to full dilation. Delivery was spontaneous, four hours and thirty-seven minutes after instillation, and with only one hard pain while head was passing over perineum. No laceration. No chloroform used. Patient drowsy for one hour following delivery. Child crying and normal.

July 22, 1923. Mrs. C. C. Para 1. Moderate size woman, having pains every five minutes and lasting fifteen seconds. One ampoule of magnesium sulphate 2 c.c. 25 per cent solution with ⅙ grain of morphin was given when cervix was dilated two to two and one-half fingers. Instillation given by syringe method when cervix was three fingers dilated, soft, but not obliterated. Whole amount retained. Two and a half hours afterwards hypodermic magnesium sulphate 25 per cent solution. Patient cooperated well. Sedative effect marked, as evidenced by sleep appearance and little pain, but patient stated she felt contractions continuing.

Some nausea and slight vomiting. Cervix dilated fully. L. O. P. position rotated to L. O. A. and was delivered spontaneously about four and a half hours after instillation. Patient had severe pains during last five or ten minutes. First degree laceration.

Conclusion.—A very good result but this patient should have had chloroform the last five or ten minutes while head was passing over perineum.

From service on the Sixth Floor (Cowan)

July 18, 1923, Mrs. V. H., primipara, large, heavy type of patient, having hard pains every minute, very noisy and complaining especially of backache which was almost unbearable. Cervix dilated two fingers. A hypodermic of morphin sulph. gr. ⅙, dissolved in 2 c.c. of a 25 per cent solution of magnesium sulphate, given. The second ampoule of magnesium sulphate without morphin was given one hour later. (Some relief but patient still complains of pain.)

Rectal instillation given one and a half hours after *first* hypodermic, cervix thin and dilated 3 fingers. At first slight irritating effect, sedative effect beginning fifteen minutes after instillation. Backache ceased immediately, pains less severe, but patient was conscious that contractions were still going on. Cervix dilated fully in forty-five minutes and delivery in one hour and forty-five minutes after instillation. Baby crying and normal in every way. No anesthetic used while head was passing over perineum, and delivery was accomplished practically with no pain—no laceration. The analgesic effect was still present after delivery, as evidenced by piercing perineum with towel clip without causing pain. No postpartum hemorrhage.

Conclusion.—Such ideal results cannot be expected in all cases.

COMMENT

It is stated authoritatively "that no drug which can so far abolish sensation as to make labor painless can be given to this degree without affecting considerably the normal process of labor." We cannot entirely concur in this statement, although it seems to be the consensus of opinion of all obstetricians and of all authorities. (As yet magnesium sulphate alone has not been tested for confinement cases.) By using the minimum dose of a number of drugs synergistically, it is believed that the above objection has been overcome. It must be remembered that when the four ounce mixture is placed in the rectum, it is still practically outside of the body.[8] When absorbed into the blood, it is only then a part of the circulatory system.

Chart used in evolving this method.

LYING-IN HOSPITAL
ANALGESIC CHART

A.P. Number..... Date.....
Formula Number.....
NAME OF PATIENT ..... Para.....

TIME	
ACTUAL	TOTAL

1. Hypodermic { with gr.... / without } morphin Cervix { Dilated....Fingers / Thick / Soft / Hard / Obliteration }
2. " effect { unchanged / sedative / exciting }
3. " repeated { without } morphin
4. " " { with gr.. / without } morphin
5. Instillation: { Retained.... / Irritated.... / Expelled.... } { Gravity... / Syringe.... } method
   " Cervix: Dilated....fingers / Thick / Soft / Hard / Obliteration
6. Effect of enema and hypodermic: { unchanged / sedative / exciting }
7. (Contractions of uterus) { Not affected / increased / decreased }

Does labor progress?

8. Sensation of pain { decreased / increased / not affected }
9. Delivery— { with forceps / without forceps }
10. Do occipito-posterior positions rotate normally?
11. Condition of baby: { crying / apnoeic / asphyxia }
12. Postpartum: { hemorrhage / contraction of uterus }

Remarks on any of above numbers on reverse side.

DIRECTIONS FOR GIVING HYPODERMIC AND COLONIC INSTILLATION.—The hypodermic should be given when the cervix is approximately two fingers dilated. If a 25% solution of magnesium sulphate is used, 1/6 or 1/4 grain of morphin may be given with the first hypodermic only; if a 50% solution, give no morphin. The hypodermic should not be repeated unless the sedative effect is wearing off or is insufficient. State object of hypodermic is to *relieve pain.* From now on the patient should be kept as quiet as possible. If in a ward the bed should be screened, if in a room lower shade and exclude light and close the door. Loud talking and noise of all kinds is avoided as far as possible. Just before giving instillation repeat object is to *relieve pain.* When cervix is about three fingers dilated accompanied by good contractions place patient on left side—Sims' position. Fill catheter with olive oil in order to exclude air and insert about four inches into rectum. Place whole amount of instillation in a syringe and give *slowly,* care being taken not to admit air between olive oil in catheter and mixture in syringe. Under gentle pressure pass the fluid *between* contractions giving the whole amount between two to four contractions—tell the patient to "squeeze up" in order to induce reverse peristalsis. State that if contents are retained there will be no pain and thus secure her cooperation. Make pressure on perineum with a towel *during* pains for 10 to 15 minutes, but withdraw tube gently in 5 or 10 minutes. From now on patient can be on back or in whatever position is most comfortable for her. Place cotton in ears and cover head with preferably some dark colored material or with a towel. Give only necessary attention to patient, talking in a low voice and making all manipulations as gently as possible. *Note time of hypodermic and instillation on special chart and fill out chart as labor progresses.*

"In 'twilight sleep,' fetal asphyxia occurs in 9.6 per cent of cases but fetal mortality is not above the average."[9] In our series the percentage is less than 1 per cent and this case was not fatal. The usual objections against twilight sleep (a) prolonged labor for hours and days, use of forceps more often necessary, the percentage of ruptured perineums higher, failure of occipito-posterior positions to rotate normally (b) restless delirium and violence, disturbance of heart and lungs, postpartum hemorrhage and uncertain results, do not seem to obtain with the present method.

In our series only in one case in 64 did asphyxia occur which gives a percentage of 0.64. As a general proposition "synergistic analgesia" is a safer condition than either oil-ether analgesia or "Twilight Sleep." In Twilight Sleep too much dependence is placed upon morphin and its action is too greatly stressed—hence the high percentage of asphyxia. With the synergistic method, we attempt to secure relaxation with the magnesium sulphate as well as using it for its power of prolonging the effect of the morphin. Ether is a powerful stimulant and analgesic as well as anesthetic. The attempt is made here to use it only for its stimulating and analgesic properties and we believe we obtain this by using it in the minimum dosage as given.

SUMMARY

1. Labor is not delayed, for while the pain is relieved the contractions continue in over 98 per cent of the cases.

2. The condition of the baby is not affected by the medication.

3. Ether administered colonically to the mother is safer from the offspring's standpoint than is sufficient morphine to produce the same effect.

CONCLUSIONS

We feel that in this small series of a little over one hundred cases we have established the fundamental principles upon which painless labor may be safely worked out, i.e., by using the minimum dose of a number of drugs, compatible and synergizing, using each drug for a definite and specific purpose.

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40 EAST FORTY-FIRST STREET.

GERMICIDES FOR USE UPON THE SKIN

By Ellice McDonald, M.D., Philadelphia, Pa.

IN July, 1915,[1] I reported the results of experimentation upon a new solution for disinfection of the hands and abdominal skin before operation of the following composition: Alcohol, 60 parts; acetone, 40 parts; and pyxol, 2 parts.

Since that time, the solution has been extensively used and many clinics have adopted it. This is particularly true of Schenectady where the paper was read and where apparently all the surgeons use it.

McMullen[2] states that "we have used the alcohol-acetone-pyxol solution in the hospital in 8125 cases during the past eight years (fourteen months of which were spent in the service) and have no infection which we felt was due to skin preparation. The number of cases which were treated before minor operation in the office is probably twice this number with most satisfactory results." Stanton[3] says that "our records show 3568 operative cases prepared by the alcohol-acetone-pyxol solution. I consider wound healing in this series almost perfect. Of course, we have had a few infections, all investigated and all traceable to causes other than skin preparation." Whiting and Slocum[4] have modified this solution by substituting phenoco 2 per cent for pyxol 2 per cent. This is a minor modification as phenoco is a germicide of the same type as pyxol and its germicidal action is apparently dependent upon the same germicidal substances. However, there is no doubt that phenoco acts very well in the solution. Four years' use of the solution with Whiting and Slocum's modification at the Misericordia Hospital, Philadelphia, shows that 3401 cases were operated upon after the skin was treated by this method. According to Geo. P. Muller,[5] the results "were eminently satisfactory."

These results substantiate the merit of the solution. In considering the question of the effectiveness of the solution, it is to be remembered that v. Herff has reported a large series of cases operated upon after skin preparation with acetone and alcohol alone, with very good results. My solution, with the addition of a strong germicide, is much more effective than that of v. Herff.[6] The burden of proof does not rest, however, upon me alone, but v. Herff's evidence is available, as well as the results in practice of the other surgeons.

If this solution had only the qualities of being a fat solvent and of containing a germicide which is not neutralized by the skin or organic matter, it would still be superior to the commonly used skin disinfectants, regardless of its high germicidal quality. These two qualities, of dissolving fat and of not being neutralized by organic matter, are absolutely necessary in a skin or body disinfectant. The coal tar germicides are the only ones not so neutralized and, of these,

it is advisable to use the one with the highest phenol (carbolic acid) coefficient.

I hold no particular brief for pyxol, except that it has a high coefficient and is not neutralized by organic matter. Cyllin with a coefficient of 14, is another preparation which might be well substituted, although I know of no one who has done so. It is to be remembered that these coal tar germicides are obtained in the preparation of other coal tar products and that they must be manufactured by being put into emulsions in various ways. For this reason, they must of necessity be made by commercial firms and, even if the essential ingredients which give them their germicidal properties be discovered, these will still require to be made available for us by being manufactured into emulsions. The germicidal substances in these coal tar germicides are insoluble in water. Their germicidal power and quality can be estimated and gauged by means of the Rideal-Walker or "Hygienic Laboratory" methods of taking the phenol (carbolic acid) coefficient and their germicidal strength can be known in terms of phenol as a unit. The germicidal value is the quality in which surgeons are interested, and this can be estimated by any properly trained bacteriologist. The law, in fact, demands that claims in regard to the germicidal value must be substantiated.

There seems, however, to be considerable misapprehension as to the relative value of germicides in surgery. Germicides are rated according to their phenol (carbolic acid) coefficient, taking phenol as the unit and testing each substance against it under standard conditions and with a standard culture, such as 24-hour B. typhosus, which has a very high resistance to germicides. This gives the ratio of germicidal value. However, it is found that some germicides, which are very efficient under test tube conditions, are much diminished in value by the presence of organic matter. This is particularly true of the heavy metals, like mercury, iodine, manganese, etc., which are active coagulants of albumin. For this reason, in all tests for surgical work, it is necessary that the germicides should be tested in the presence of organic matter in order to approach the conditions of practice as much as possible. In body disinfection, organic matter must always be present in excess.

Germicides may be roughly divided into chemical germicides and emulsion germicides. The chemical germicides act by combining in direct chemical combination with the protein of the bacteria and so destroying them. These chemical germicides, at the same time, combine with any other protein substance present, such as the body tissue, blood, pus, serum, soap, etc. For this reason, all chemical germicides, while destroying bacteria, at the same time destroy tissue. They are usually applied in strengths which will cause but little tissue reac-

tion, but the destruction of tissue is there whenever they are applied to the body. An example of this type of action is the destruction of tissue caused by silver nitrate, bichloride of mercury or iodine. The chemical germicides are always neutralized by organic tissue and to be effective must be present in excess, as in the Carrel-Dakin method, but, even so, their action is limited by the barrier of altered tissue which they themselves form in combination with the skin or other body surfaces. Some of the chemical germicides stain the skin and this is considered proof of their action. As a matter of fact, the staining of the skin shows that they enter into chemical combination with the organic matter of the skin and is a proof of the limitation of their action. If this limited action is all that is required, there is nothing more to be said; but, if continued germicidal action is needed, it can be best obtained by germicides which are not neutralized by organic body tissues.

The emulsion germicides, on the contrary, are substances which are insoluble in water and are suspended in emulsion by various means, such as soap, gelatin, etc. The action of these upon bacteria is not fully understood, although the results of such action can be readily tested and gauged by taking their phenol coefficient. It may be that they take on some of the qualities of colloid substances. Why a substance, which is insoluble in water and does not combine with protein, does kill bacteria, is difficult of explanation, but tests of the phenol coefficients of these coal tar emulsions show that they are often very strong germicides, and, as a rule, are not neutralized by organic matter. In fact, when a substance can be put into both solution and emulsion, it is usually much more efficient germicidally in the emulsion. The germicidal action of these emulsion germicides is not the result of chemical union (as is the case of the heavy metals, formalin, etc.) but is associated with the emulsification of the colloidal suspension, as evidenced by the precipitation of proteins when a certain phenol concentration is reached. This action seems to be similar to heat coagulation. It is for this reason that the higher strengths of phenol disinfectants are proportionately stronger than the weaker strengths, as, for example, phenol (carbolic acid) below 1-200 becomes disproportionally weaker germicidally.

Most of these coal tar germicides are described as higher phenols or phenol homologues for want of a better name, but this does not mean that they have any of the character or poisonous qualities of carbonic acid. It is probable that there are a number of substances in them which have high germicidal qualities, as can be proved by the phenol coefficient. The one which has the highest germicidal value is thought to be para-hydroxi-diphenyl. This substance has been isolated in crystalline form and is insoluble in water. An emulsion of it has been

prepared experimentally and has a coefficient of 40 and is apparently but slightly toxic. At present, it is only produced experimentally, but it will make an ideal surgical germicide and would be an improvement in the solution here described.

The chemical germicides are all neutralized by organic matter. Bichloride of mercury, for example, is the best germicide under test tube conditions and without organic matter, as it has a coefficient of over 200*: but this coefficient falls to 20 with the presence of 1 per cent of organic matter, and, with further additions, the salt is neutralized and the germicidal action gone. Without organic matter, this salt will kill anthrax spores in 2½ minutes in the proportion of 1-3000, being one of the few germicides which will do this; but, with organic matter, it will not kill anthrax spores, even in a dilution of 1-40. This shows the uncertainty of the heavy metal germicides.

Pure iodine, without organic matter, has a coefficient of 38, but, with 1 per cent organic matter, this is reduced to 7, and, with further additions, the metal is neutralized and the germicidal action gone. It is obvious that iodine on the skin forms a chemical combination with the outer layer of the skin and that then the iodine is neutralized. The outer layer is rubbed off or washed off during the operation and the unstained skin is left. It is probable that weak solutions of iodine, such as 2 per cent, are as efficient germicidally upon the skin as stronger ones, as there is no more than a superficial disinfection in any case. This is well shown by Robb who removed the excess iodine from the skin by potassium iodide in order to remove its inhibiting action in cultures and was able to recover bacteria from the skin surface in from 50 to 95 per cent.

If iodine is used upon the skin, it should be combined with a fat solvent in order to penetrate the glands and dissolve the fat. Before my present solution was devised, I published,[8] in 1911, the description of a carbon tetrachloride-iodine-two-per cent solution which can be rubbed in the skin and contains a good fat solvent. The mechanical assistance given a germicidal solution by being able to rub it into the skin must be considerable help in getting disinfection. Alcoholic solutions of iodine do not penetrate the fat of the skin and how can disinfection take place when the fat of the sweat glands and hair follicles is not penetrated? Alcohol is not a solvent of fat as found in the skin, i. e., the glyceride. The sole glyceride that alcohol will dissolve is castor oil and this action of alcohol is so unique that this is a test for castor oil; but, upon the skin glycerides, alcohol has no action.

One very interesting action of iodine upon the skin is the delay of healing which follows its use. Stanton[7] has emphasized this chemical

*The coefficients here described are by the R. W. Method.

delay of healing and estimates that it often amounts to several days and is dependent upon the strength and application of the iodine solution. The delay seems to occur because there is tissue destruction which must be overcome and eliminated before true healing can begin. This is one argument for weaker iodine solutions as the irritating and blistering action of the stronger solutions is well known. Iodine was used as a counterirritant long before its use as a skin disinfectant.

Iodine does not seem to have any advantage over bichloride of mercury as a skin disinfectant. Both are irritating and both are neutralized by organic matter. Bichloride of mercury is a much stronger germicide, and it is apparently not any more irritating than iodine. However, the staining of the skin by iodine seems to make a deep impression and gives psychological comfort to the surgeon. Germicides which stain seem to be very appealing, as is shown by the recent vogue of picric acid which has a coefficient of only 2.5.

Hypochlorite of soda has a coefficient of 0.6 without organic matter, but, with organic matter, this coefficient is reduced to 0.075, both of these coefficients so weak as to be negligible. It is obvious, therefore, that the effect of Dakin's 1-200 solution must depend upon antiseptic action which is a very much different quality from germicidal action.

Potassium permanganate has a coefficient of 50 without organic matter, but, with organic matter (gelatin 1 per cent, serum 1 per cent) this is reduced to 7, and, with more organic matter, the salt is neutralized as a germicide, as is sodium hypochlorite. It would seem, however, that potassium permanganate has been neglected as a disinfectant for continuous wound irrigation as 1-10,000 permanganate would be theoretically equal to Dakin's solution in germicidal value. This is a rough comparison and true within limits. The ease of making the permanganate solution would be a great advantage and might overcome its staining properties. Permanganate is certainly worthy of more use in open wounds and in continuous irrigation where it is used in excess and frequently renewed.

Formaline has a coefficient of 0.3 and combines with organic matter. The germicidal value of this substance is so weak that it has no place in surgical work for any disinfectant purpose.

Picric acid in alcoholic solution has recently come into vogue as a skin disinfectant. This substance has a coefficient of 2.5 without organic matter. The 2 per cent alcoholic solution would thus be one-tenth as germicidal as my solution, or, in other words, my solution is ten times as germicidal. Picric acid, in addition, is neutralized by organic matter.

Alcohol as a germicide is so weak as to be not worth considering. This has been proved so often by so many observers that recapitula-

tion would be wearisome. I would refer any interested to Harrington's article. An evidence of the weak action of alcohol is that wines, such as sherry, must be fortified to 18 per cent alcohol so that they do not decompose. The belief in alcohol is largely traditional and is a relic of the conviction that alcohol was good for any ailment. It has, however, a useful solvent power upon substances other than fat. Its dehydrating action makes it of use when combined with acetone.

Quinine is a much neglected germicide; the bisulphate has a coefficient of 3. The antiseptic action of quinine is very high. According to my experiments, it inhibited growth of fresh typhoid culture in the strength of 1-30,000. Upon the same culture, bichloride of mercury inhibited growth in the proportion of 1-50,000 and pyxol in 1-20,000. This antiseptic action of quinine may be made use of in wet dressings of raw wounds or infected surfaces in the proportion of 1-1000 with boric acid. Either the bisulphate or the bichloride may be used. Quinine bisulphate is also useful for bladder cystoscopic work or bladder irrigations in the proportion of 1-1000 as described in a former paper.

Some other coefficients of interest to surgeons are: creolin 2.5, benzoic acid 4, salicylic acid 6, eucalyptus 1.2, lysol 2.5, guaiacol 1, thymol 20, cyllin 14, izal 10, phenoco 15, pyxol 20, etc.

Antiseptic action is apparently dependent upon different qualities from germicidal action. Inhibition of growth is sometimes produced by substances which have weak germicidal qualities, such as quinine. It may be that such antiseptics have a special action upon microorganisms by preventing their reproduction or else upon the early budding stage. Organisms have varying powers of resistance, so that some are more easily killed, B. typhosus for example, being very resistant. Resistance is, in my opinion, constant for any germicide that has sufficient action to be measured by the phenol coefficient. This is the only accurate method of judgment for any germicide for surgical purposes and, if the germicide is not of sufficient strength to be measured, it is of no use in surgery.

It is questionable whether the destruction of spores upon the skin surface is ever possible. Bichloride of mercury, the strongest germicide known, will not kill anthrax spores in the presence of organic matter in 1-40 solution. Spore-bearing bacilli which cause disease are anthrax, tetanus and gas gangrene. In my opinion, no chemical preparation will kill these spores in the presence of organic matter within the limits of time required for surgical preparation. Dependence must be upon the mechanical solvents of the solution and this is one of the advantages of being able to rub a solution into the skin. Spore-bearing bacilli are fortunately very rare upon the skin. Maher, in fact, denies that they are ever found, except in obviously diseased

skin. But, in any case, it is better to depend for their destruction upon a solution which is not neutralized by organic matter and which acts over the period of several minutes that it is applied, than upon a solution which is immediately neutralized by the skin itself.

It must not be thought that it is claimed that my solution is stronger than iodine under test tube conditions and without the presence of organic matter. Under these conditions, the commonly used iodine solution is probably about three times as strong as my solution, but disinfection of the skin is an entirely different problem from disinfection without organic matter. On the skin, iodine is promptly neutralized, while the coal tar germicide acts during the time it is applied and this prolonged action, due to the fact that it is not neutralized, makes its weaker germicidal power more effective. Iodine in the proportion of 1-10,000 will kill staphylococcus very promptly without organic matter, but, in the presence of serum, iodine in 1-800 dilution will not kill staphylococcus within half an hour.

Epidemics of infection in clinics are seldom due to faulty skin preparation. It is the isolated case which is due to skin infection. If one clean case after another becomes infected, it is reasonable to suppose that the infection has been introduced from without from some infective focus, elsewhere than in the patient's skin. It would be improbable that each clean case harbored the same germs of infection; it is more probable that in epidemics, there is a common source of infection, outside the patients' skin. For this reason, in epidemics of infection, a source or focus outside the skin should be searched for. Sore throat among the operating room attendants is a common cause and Stanton[9] and McMullen have reported a series of infections from this cause. The operating room staff should be instructed to report any sore throat or other infective focus which they may harbor, so that they can be eliminated from the operation.

No doubt, many surgeons will continue to use iodine for the skin and, if they do so, a fat solvent should be combined with the iodine as in my carbon tetrachloride-iodine-two-per-cent solution.[8] The same effect is by no means obtained by preceding the alcoholic iodine by a fat solvent. The fat solvent should carry in the iodine.

However, for hand disinfection, iodine is not used on account of its irritating qualities. Here, the coal tar solutions have no rivals as they are not irritating to the hands and they are not neutralized by the preceding soap. Washing the hands with soap and water alone has no disinfectant value. This has been repeatedly proved. However, surgeons are not likely to give up washing their hands before operation, so that it might as well be made of some use by combining, with the wash water, a germicide which is not neutralized by soap, so that the water may have some germicidal value and the hand wash-

ing be to some purpose. This germicide can only be one of the coal tar germicides and the fact that they are emulsions, aids their effectiveness in wash water with soap.

The following directions in the use of my method are given as it is very commonly misunderstood.

DIRECTIONS FOR DISINFECTION OF THE HANDS (MC DONALD METHOD)

As washing the hands with soap and water has no effect upon the bacteria of the skin, even if kept up for 15 minutes, it is advisable that some germicide should be added to the wash water. This germicide should be one which is not neutralized by soap or organic matter as contained in the skin, such as pyxol 1-1000 (or phenoco, if preferred) in the basin of warm wash water. This strength will kill B. typhosus in 2½ minutes and the pus forming organisms are more easily killed than B. typhosus.

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The area under the nails is the most difficult place to disinfect and, therefore, particular attention should be given to the introduction of the germicidal solution under the nails. Nail brushes should be used for the nails only and wash cloths for the hands and arms.

1. Wash the hands in soap and water, containing pyxol 1-1000 (or phenoco 1-1000), in a basin for three minutes. Scrub the nails with a nail brush and the hands and arms with a cloth. This solution may be used repeatedly. This thorough scrubbing is done only at the first operation and, after this, the time may be reduced to two minutes.

2. Keep wooden orange sticks and nail files in McDonald's solution and do the toilette of the nails, making an effort to introduce the solution under the nails by means of the wet nail file and orange stick.

3. Hands should then be washed in McDonald's solution for three minutes scrubbing the nails with the nail brush and using a cloth for the hands and arms. The solution should be at least three inches deep in the basin. The hands are then dried and prepared for gloves.

Preparation of Patients.—Shaving the night before operation should be done with water containing pyxol 1-1000 (or if preferred phenoco). In this way, the shaving water will be a germicidal solution and will aid disinfection, and, as the skin is again disinfected the next day, fractional sterilization will be practiced. At operation, the dry towel is removed and the area of operation rubbed lightly with a small towel or piece of gauze saturated with McDonald's solution, for three minutes. At the end of this time, the wet towel is placed over the operation area and allowed to remain there until the operator is ready when it is removed and the skin lightly dried.

In preparing for vaginal operation, the patient should have a douche of pyxol 1-2000 (or phenoco) the night before operation and the vagina washed at the time of operation with soap and water contain-

ing pyxol 1-2000. In this way, the water will have some germicidal value which it does not have with plain soap and water.

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8305 SEMINOLE AVENUE.

DYSTOCIA IN AN ECLAMPTIC DUE TO A CASE OF CYSTIC HYGROMA OF THE PECTORAL REGION OF THE FETUS

BY FRANK T. IIAMS, M.D., HOUSTON, TEXAS

I DESIRE to place on record the following case of a cystic hygroma of the pectoral region, in a fetus, large enough to cause dystocia, which is rare, and I hope of sufficient interest to justify its presentation.

Patient a primipara, colored, housewife, 19 years old. Mother, father, and three brothers and sisters, all living and well. No defectives.

Has been married one year. Husband living, has a two plus blood Wassermann and irregular attacks of asthma.

Patient healthy all her life, except for usual childhood diseases, and a leucorrhea for the past year. Menstruation regular, 30 day type, five days, moderate flow, no pain, last period February 6, 1922, due about November 13, 1922.

History of present illness: Nocturia, 4 or 5 times and edema of the feet and legs for past three months. Constipation, dizziness, and frequent blind spells for past week.

On November 11th, 1922, patient was admitted to the Houston Municipal Hospital, in a semicomatose condition, after having been in labor for 18 hours, attended by a negro doctor. She was seen immediately by the interne, who reported that she was having severe pains every minute, that the cervix was soft, thin and four fingers' dilated, membranes ruptured, blood pressure 210 over 130, which rose to 225 systolic during a pain, and that the fetal heart was 160 and irregular. Urine showed a heavy cloud of albumin.

The interne was advised to take the patient to the delivery room and completely dilate the cervix under ether anesthesia, apply forceps and deliver as quickly as possible. Upon my arrival some thirty minutes later the head was on the perineum. The interne said that the forceps had slipped twice, they were reapplied, proper pull instituted and the head was born in the occiput posterior position, but no further progress could be made.

The pelvic measurements were normal. The uterus was firmly contracted upon the fetus and no fetal heart sounds could be heard. Maternal pulse was almost imperceptible and the patient was semicomatose.

A small fetal head and a uterus well filled up made one think of twins complicated by contraction ring, or of a monster. Vaginal examination revealed an

unusual cystic condition on the back of the fetus, which gave the sensation of a hematoma. After a consultation, embryotomy was decided upon. The sac referred to was punctured and about one pint of fluid was removed, but still the uterus held the fetus. After decapitation, the removal of an arm, and the removal of the left lung, it was decided to risk an anesthetic. When the uterus relaxed a cystic condition was found anteriorly, which was punctured and about one quart or more of clear straw colored fluid was withdrawn and finally the fetus was extracted.

A second degree laceration was not repaired and the puerperium was stormy, being complicated by low grade peritonitis, pyelitis and phlegmasia alba dolens. Patient left the hospital January 7th in good condition.

Fig. 1.

Inspection showed a female child at term, apparently fully developed, with a large tumor mass on the left side of the thorax, extending laterally, from flexor surface of wrist to costal margin; anteriorly, from sternum and lower surface of clavicle to vertebrae and upper part of scapula posteriorly. The arm was nearly encircled by the tumor mass, except for a narrow strip on its posterior aspect. The surface of the tumor anteriorly was irregular and appeared somewhat nodular, elsewhere the skin surface was smooth. The left nipple was displaced 7.6 cm. from sternum and 7.6 cm. lower than right nipple. The areola was about five times larger than the right areola and had several small locules in it.

Approximate measurements were as follows:—length, 48 cm., abdominal circumference, 27.5 cm., greatest circumference of tumor around thorax 58 cm., greatest circumference of tumor taken vertically 40.5 cm., cord-length 60 cm., Placenta 19x16.5x3.5 cm.

Autopsy showed all internal organs normal. Incisions in tumor mass disclosed a multilocular cyst, containing clear, straw colored fluid, with 20 or more locules, varying in size from a pea to a hen's egg, each as a rule communicating with one or more locules, with some locules entirely separate. The tumor anteriorly was old, while that posteriorly was comparatively new, which carries out the idea that these so-called neoplastic conditions (and it is doubtful whether they should be placed among true tumors) spread along the lines of least resistance as more and more of the fluid accumulates. In this case the fluid had accumulated between the skin and subcutaneous tissue in front and the pectoral muscles behind on the left side. There was an extension of the cavity upward under the lower margin of the pectoralis major for a distance of little more than a centimeter. In tracing the cavity outward and downward along the left arm the coracobrachialis and biceps muscles came into view. On the posterior aspect of the left shoulder we were able to trace an extension of the cavity under the trapezius muscle, and in this region the deltoid and a part of the posterior scapular group of muscles came into view as forming a part of the anterior wall of the cyst. Microscopic sections of the walls of the cyst reveal varying thicknesses and to be made up of fibrous tissue, while the inner walls were found to be lined in places with an endothelial layer.

Comparatively speaking, cystic hygroma is a rare condition. In the Annals of Surgery for July, 1913, according to Dr. Chas. N. Dowd of New York, only 91 cases of cystic hygroma of the neck had been reported in the literature previous to his article, 35 cases of cystic hygroma of the axilla and 11 cases in other portions of the body. Dowd reports four cases. Since his article appeared other cases have been reported by Matthews, Downes, Lyle, Winslow and others; Winslow reporting a case of the pectoral region in a child 4½ years old.

Even though the condition is congenital it may not be noticed at birth. Occasionally at birth it is thought of as a hematoma and later as a nonpainful, harmless mass. It is a condition that usually manifests itself in infancy, less frequently in childhood, and rarely in adult life. Sometimes its presence is noticed as a harmless mass throughout life. One of the chief characteristics is its rapid growth along the line of least resistance and it often envelops important vessels and nerves, making operative treatment hazardous.

807 KRESS BUILDING.

TUBERCULOUS OVARIAN CYSTS WITH A REPORT OF A THIRD CASE OBSERVED BY THE AUTHOR*

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TUBERCULOSIS of the female genital tract is a common disease and is found especially in the uterus and the tubes. The ovary, on the other hand, seems to be fairly immune as numerous examples and statistics show. Schlimpert found the ovary attacked in only 4.1 per cent of the cases of genital tuberculosis. v. Franqué reported an instance where the whole genital tract and the peritoneum were tuberculous but the ovaries were perfectly intact, despite the fact that the infection was of sixteen years' standing. Martin could find only 173 reports of tuberculosis of the ovary in 1899. However, some authors give much higher figures although usually based on rather small series. Cummins, for example, claims that 34 out of 40 cases (85 per cent) of genital tuberculosis examined by him showed involvement of the ovary. These figures are so high that I strongly suspect that they include not only ovarian tuberculosis but also tuberculous peri-oophoritis. The percentages given by other men vary very much and are as follows: Wolff 60 per cent; Terillon 50 per cent; Williams 44 per cent; Kelly 33.3 per cent (in children); Greenberg 33.1 per cent; Orthmann 33 per cent; Oppenheim 33 per cent; Labhardt 32 per cent; Neu 31.7 per cent; Berkeley 22.5 per cent; Ollivier 17 per cent; Rives 15 to 20 per cent; Kroemer 15.7 per cent; Horizontow (from the statistics of Bollinger, Schmorl, Schmaus, Ribbert and Albrecht) 15.4 per cent; Merletti 14.5 per cent, and Spaeth 12.6 per cent. A number of these authors also state that both ovaries are usually affected.

Judging from the records of the pathological laboratory of the New York Post-Graduate Medical School and Hospital, ovarian tuberculosis certainly is not common, as only seven such cases—mostly unilateral—have been seen there since 1913. In my own experience also, ovarian tuberculosis has been very rare. For a number of years I was in a locality where genital tuberculosis was very frequent, yet despite this fact, I did not see over a half dozen cases of ovarian tuberculosis and not a single bilateral infection. At the same time

*Read at the meeting of the N. Y. Path. Soc., on March 14, 1923.

it must be admitted that genital, peritoneal, or even pulmonary, or bone tuberculosis is at times associated with cystic formation in the ovary. These cysts themselves, however, are practically never tuberculous although a simple inflammatory reaction is often present. Nevertheless, some authors claim that the tuberculosis, by means of toxins, or otherwise, is still the direct cause of such cysts. These men consider the menstrual disturbances so frequent with tuberculosis as a further visible proof of their theory. Pollosson and Violet and Poncet and Leriche are especially fervid adherents of this tubercular etiology of many ovarian cysts. Poncet even goes so far as to assume that the inflammatory reaction of the ovaries in such cases is a genuine tuberculosis that has somehow lost its characteristic histological appearance. He calls this condition "Tuberculose Inflammatoire."

Although I have admitted just now that ovarian cysts are at times found in conjunction with local or even general tuberculosis, I must say that I can see absolutely no reason for accepting a casual relationship. Coincidence may play a large part in such findings, in fact it would be strange if two such common ailments as tuberculosis and ovarian cysts were not sometimes combined. Furthermore it is not very clear anyhow why tuberculosis should be more conducive to the production of ovarian cysts than any of the other inflammations. Personally, from my own experience and after a survey of the literature, I cannot believe that ovarian cysts are really so common with tuberculosis as Pollosson and Violet and Poncet and Leriche and their followers would have us think. Simmonds, for example, to mention only one name, found 80 cases of genital tuberculosis among 6000 female cadavers. Only 4 of these 80 cases showed ovarian cysts and Simmonds mentions expressly that the cysts were of long standing and certainly only accessory lesions. Not one of the 4 cysts showed a tuberculosis of its wall or contents. This absence of tuberculosis in ovarian cysts found associated with definite tuberculous lesions in the rest of the genital tract is another factor which speaks strongly against a tuberculous etiology for such growths, unless indeed one wants to accept Poncet's *tuberculose inflammatoire* as a genuine tuberculosis. This, however, does not seem warranted because it would then be difficult to explain why the tuberculous lesions in these organs should be totally different from such as are found anywhere else in the body. Furthermore, typical tuberculosis does at times occur in ovarian cysts so that it cannot be said that the ovarian tissue itself reacts differently from other tissues to the tuberculosis bacillus. Such cases of tuberculosis of ovarian cysts are, however, rare.

Schottlaender in 1892 could find only 7 cases of tuberculosis of ovarian cysts. Pruesmann in 1904 found 6 more. Celler later in the same year collected 20 cases including his own. A closer investiga-

tion caused Celler to eliminate 7 of the cases because they either were not examined properly or were not neoplasms. Among the seven was the case of Madlener which should not have been eliminated as it really was a tuberculosis of an ovarian cyst, a fact which has since been brought out by Madlener himself. Up to 1904 then there were only 14 positive cases of tuberculous ovarian cysts reported in the literature. Since then a few more have been reported sporadically.

In 1915 I was able to report the first case of my own.

This was a nulliparous, single woman, 50 years of age (M.L.M. Chart No. 403, 1915, Frauenklinik, Tuebingen). She was generally asthenic, had lost much weight and complained of pains in the abdomen, also of cough.

The physical examination showed pulmonary tuberculosis and a left sided ovarian cyst as large as an adult head and a right sided cyst about the size of a goose egg. At operation, under spinal anesthesia, a general peritoneal tuberculosis with many adhesions, but no ascites, and the described cysts of the ovaries were found.

Because of the poor condition of the patient only the adnexa and a piece of peritoneum were removed. Pathological examination showed that the large cyst was of serous type, the smaller one a dermoid. Both the peritoneum and the wall of the large cyst were tuberculous and numerous tubercle bacilli could be demonstrated in the sections. Both tubes and the dermoid were perfectly free from tuberculosis.

An interesting point in connection with this case was the fact that the woman developed marked ascites shortly after the operation. No doubt the traumatism from the operation which was rather severe, due to the many adhesions, produced this ascites. Frequently a simple laparotomy will improve cases of peritoneal tuberculosis but, on the other hand, marked trauma may also cause an exacerbation of the tuberculosis; an accident which I have seen also in other cases of genital tuberculosis of the female and to which I have called attention in another place.

About six months after the operation the patient died of cachexia. An autopsy revealed an atrophied uterus free from tuberculosis.

In 1916 at Tuebingen I saw a second case of tuberculous ovarian tumor. This time it was a solid adenofibroma of the left ovary with a tuberculous infection. This is the only instance in the literature of a solid ovarian tumor infected with tuberculosis with the one exception of Glockner's case where a medullary carcinoma and tuberculosis were found together.

This second case (R.K.J. Chart No. 719, 1916) was a sterile married woman of 43 who had had pelvic and abdominal pains for a long time.

Physical examination showed a large fibromyomatous uterus and large bilateral masses.

At operation, also under spinal anesthesia, marked perimetritis and double pyosalpinges were present. Both ovaries were firmly adherent to the tubes, the right one small, the left one firm, solid, about the size of a small orange. The peritoneum was not diseased. The uterus, both tubes and the left sided tumor, which was a fibro-adenoma as mentioned above, were removed and microscopical examination proved them to be tuberculous. The right ovary, which was also removed, was small, fibrotic and free from tuberculosis. Tubercle bacilli were again found in the sections from the ovarian tumor.

In connection with my two cases I went over the literature on this subject very carefully and found altogether 29 cases including my own, of proven tuberculous ovarian neoplasms. These were the following: 1. v.Baumgarten; 2. Bertino; 3. Brons; 4. Celler; 5. Elsaesser; 6. Gade; 7. Glockner; 8. Gruenbaum; 9. Kelly; 10. Klebs; 11. Lagothetopulos; 12. Madlener; 13. Meriel; 14 and 15. Moench; 16. Neumann; 17. Olshausen; 18 and 19. Pollosson and Violet; 20. Pruesmann; 21. Rosenthal; 22. Saenger; 23. Scalone; 24 and 25. Schottlaender; 26 and 27. Todorsky; 28. Wechsberg; 29. Spencer Wells.

In this research I overlooked three cases as I found out later, namely, those of 30. Tusini; 31. Charbonnel and Nadal and, 32. Cohn. Since that time I can find only a single case, that of Forgue and Chauvin in the literature.

Among these 33 tumors 7 were intraligamentous, about a dozen were dermoids, and only 2—Glockner's case and my second one—were solid growths.

I realize that in many articles on this subject more cases have been reported than I have just named, but all of these so-called tuberculous ovarian cysts will not stand close investigation. Forgue and Chauvin for instance have left out many certain cases and included others which are not tuberculous cysts at all, so for example, the 4 cases of Simmonds mentioned in the beginning of this paper. Miss Gorowitz is another author who does not sift her material. As early as 1899 she claimed to have found 19 cases of tuberculous ovarian cysts although she only enumerated 10. The other 9 no one has ever been able to trace. Pruesmann—as mentioned—only discovered 14 such cases in 1904.

The following have all been cited as tuberculous ovarian cysts but are in reality either tuberculous abscesses, infected follicles, corpus luteum cysts, or infected peritoneal sacculations: 1. Bouget and Albertin; 2. Edmunds; 3. Ehrendorfer; 4. Goullioud; 5. Griffiths; 6. Grusdew; 7. Heiberg; 8. Hofmeier; 9. v.Krzywiki; 10. Sturges; 11. Mangold. Remy has also eliminated the following in addition to the

above: 12. Brouardel; 13. Schreiber; 14. Jacobs; 15. Wolff; 16. Phocas; 17. Schwartz. Rives, Guillemain, and Mangiagalli as 18, 19 and 20 must also be excluded.

To these 33 cases of tuberculous ovarian neoplasms I can now add a thirty-fourth which I saw recently at the New York Post-Graduate Hospital.

Dr. Ward J. MacNeal, Director of the Departments of Pathology and Bacteriology at the Post-Graduate Hospital, first called my attention to the pathological specimen. The woman was a private patient of Dr. H. Dawson Furniss and I want to thank both of the doctors at this time for their courtesy which enables me to present the case here.

The history is as follows: Mrs. R. P., admitted Dec. 5, 1922, to the Post-Graduate Hospital. Chart No. 26431, Pathological No. 7064-14065. She was an Italian woman 29 years old who had been married ten years and had never been pregnant. Her family history was negative.

Present history: The patient had been well, according to her own statements until August 12. At that time she had pain in the left adnexal region and from August 15 to August 18, 1922, a slight uterine flow. After that her menstruation—which before had always been regular—ceased. Her abdomen enlarged and she had nausea and vomiting lasting about 3 weeks.

Physical examination showed no general abnormalities. The abdomen, however, was enlarged and tender on the left side.

Gynecological examination revealed a large mass on the left side which could not be mapped out distinctly. It extended almost up to the umbilicus and projected below into the true pelvis. The culdesac of Douglas was filled out and felt more or less doughy. The uterus could not be isolated very well but seemed enlarged and irregular. A diagnosis was difficult but apparently a fibromyomatous uterus and an ectopic gestation were present.

At operation, under ether anesthesia, the uterus was found slightly enlarged but otherwise normal. The mass on the left side was a large pyosalpinx and an ovarian cyst the size of a lemon. On the right side a smaller pyosalpinx and a normal sized ovary were present. The adnexa of both sides were removed. The operation was extremely difficult due to many old firm adhesions to all the surrounding structures. The peritoneum itself was not diseased.

PATHOLOGICAL EXAMINATION OF THE SPECIMEN

Gross examination: The left tube and ovary, bound up in an inflammatory exudate and fibrous tissue, measured 110 x 63 x 64 mm. At one point the specimen had been torn and showed an irregular cavity 40 x 35 x 20 mm. which was lined by a hemorrhagic pyogenic

membrane. On section it was found that the cavity narrowed down on one side to 18 mm. and was continuous with the lumen of the tube. The walls of the latter were very markedly thickened by edema and inflammatory fibrous tissue.

The ovary itself measured 80 x 51 x 45 mm. It was rather soft. On section a cavity 45 x 35 mm. was filled with opaque greyish yellow grease and numerous blonde hairs. The latter had their origin in part in the smooth wall of the cyst. Other structures such as teeth, etc., were lacking. The average thickness of the cyst wall was from 3 to 5 mm. At one end the ovarian tissue was 30 mm. thick, here a second smaller cyst filled with sebaceous material, was found.

The right tube and ovary also were bound together by adhesions especially at the fimbriated end which was closed. The tube itself measured 105 mm. in length and varied in thickness from 8 to 25 mm. Its lumen was filled with opaque yellowish grey muco-pus. The wall was 5 to 14 mm. in thickness. The ovary measured 58 x 45 x 27 mm. On section it was edematous and contained numerous follicular cysts up to 35 mm. in diameter.

Sections were cut from the various parts of the specimen; they were hardened in formalin; embedded in paraffin and strained in the usual manner with hematoxylin and eosin.

Microscopical examination: Both tubes showed a huge chronic purulent inflammation throughout and in addition, in many areas numerous typical epithelioid-cell tubercles containing Langhans giant cells.

The sections from the left ovary presented an even more advanced picture of tuberculosis than the tubes. There were many tubercles throughout the ovarian stroma. The described cyst was lined by stratified squamous epithelium, beneath which there were abundant sebaceous glands and a few hair follicles. In direct proximity to these and the cyst wall itself there were again many tubercles.

The sections from the right ovary were unimportant except for numerous typical follicular cysts and a fair number of developing graafian follicles. Tubercles were not seen anywhere in this ovary.

Smears made from the tubes, stained by carbol-fuchsin and decolorized in acid alcohol and absolute alcohol, showed numerous thin, often kinked, red bacilli.

Diagnosis: Bilateral tuberculous salpingitis.
Dermoid cyst and tuberculosis of left ovary.
Small cystic degeneration of the right ovary.

Here again, as in all the other cases of ovarian tuberculosis I have seen, the tuberculous process was present in only one ovary.

In closing I would like to say a few words regarding tuberculosis of ovarian cysts in general. I do not believe as Poncet and others

would have it, that tuberculosis is especially prone to cause ovarian neoplasms. Aside from the reasons already given it seems to me that if Poncet were right, bilateral tumors should be the rule instead of the exception. Secondly, genital tuberculosis is very often associated with thrombosis of the ovarian arteries. A thrombosis of the ovarian arteries, however, certainly would not only not be conducive to new growths in the ovary, but indeed would tend to cause cysts, which had already formed, to regress, due to the diminished blood supply. I do not believe however that anyone can decide definitely in cases of tuberculous ovarian cysts whether the tuberculosis was first or the ovarian tumor. Instead of the tuberculosis causing the cysts it is just as feasible to suppose the ovarian cysts had caused a *locus minoris resistentiae* which would allow the tuberculosis to gain a foothold.

The mode of infection in these cases must be either hematogenous or by direct contact, that is, through the lymph channels. In my first case mentioned in this paper, a peritoneal tuberculosis was present but no tuberculosis of the tubes or uterus, so that a direct extension from the peritoneum to the ovarian cyst seems probable. A hematogenous infection certainly ought to have attacked the tubes also as they are so much more sensitive to tuberculosis. In the second and third cases no peritoneal tuberculosis was present but the tubes in each case were severely infected so that here too I believe we can think of an involvement of the ovarian tissue from the tubes through the lymph stream. Another possibility is that the tuberculosis was primary in the ovaries. This, of course, cannot be proven but does not seem probable in view of what has been said before.

In addition to the hematogenous, or the contact or lymph stream infection, a direct implantation of the tuberculosis may follow the tapping of an ovarian cyst accompanied by tuberculous peritonitis. Naturally such cases were much more frequent in former years when large ovarian cysts used to be tapped as a routine. A few cases of tuberculous ovarian cysts which had been tapped and showed a tuberculosis of the wall or contents have indeed been explained in this way. Nevertheless such obvious traumatism is not needed in every case and spontaneous rupture of the tuberculous process into the interior of an ovarian cyst may well occur.

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Society Transactions

TRANSACTIONS OF THE AMERICAN GYNECOLOGICAL SOCIETY

FORTY-EIGHTH ANNUAL MEETING

HOT SPRINGS, VIRGINIA, MAY 21-23, 1923

DR. THOMAS J. WATKINS, Chicago, Ill., read a paper entitled **Cystocele and High Rectocele.** (For original article, see p. 389.)

DISCUSSION

DR. FREDERICK C. HOLDEN, NEW YORK CITY.—Cystocele, a simple lesion easily cured. I take it that a few years ago such a statement would not have passed unchallenged.

During the last decade there have been several interesting and more or less satisfactory operations proposed for the cure of cystocele and rectocele by members of this Society. The problems involved have been the etiology, the anatomy of the parts involved, and the operative procedures for the cure of the defects. Dr. Watkins' very sane conclusions as to the etiology, and his very convincing presentation of the subject, would lead one to believe that he has justified the title of his paper. Those of us who were in Chicago recently were very much interested in the presentation at his clinic at St. Luke's Hospital; so much so that upon my return to New York I started to familiarize myself with the steps of the operation there demonstrated, and thus far I have been so pleased with the satisfactory results that I am led to believe that when I perform this operation with the precision and refinement of technic as demonstrated by Dr. Watkins, I will then have an operation as satisfactory in my hands for the younger women, as is the interposition operation in women past the childbearing period.

Those of you who have not been to Chicago and seen Dr. Watkins demonstrate this operation have yet in store one of the most delightful treats you have ever had.

DR. WILLIAM C. DANFORTH, EVANSTON, ILLINOIS.—I have met the problem of the high rectocele in a manner a little different from that described by Dr. Watkins. A woman presented herself with a high rectocele coming from the cervix down, and rolling out the entire perineum and rectum. I have dissected up to the level of the cervix, freeing the vaginal wall from the rectum, extending dissection up behind the cervix. Then by passing sutures through the upper part of the levator on one side, through the posterior surface of the cervix and through the levator of the opposite side, the levators and cervix were made to form a continuous wall which retained the prolapsing bowel.

DR. WILLIAM E. STUDDIFORD, NEW YORK CITY.—In reference to high rectocele, I think there are two types of cases, one in which we have an elongation of the culdesac in which the culdesac of Douglas pushes down the posterior vaginal wall, causing a real hernial sac, and often that sac is filled with loops of small intestine and the protrusion comes down posterior to the cervix.

There is another type of case in which the culdesac does not descend as far, but we have a protrusion of the anterior wall of the rectum and posterior wall of the vagina. The handling of these cases operatively is a little bit different. Where Douglas' pouch comes down I follow high dissection as Dr. Watkins has described, but in a little different way. I start a midline incision at the cervix and make a dissection of the mucous membrane, opening the culdesac, so that the peritoneum is exposed, and with the finger it is possible to find out the amount of that descent. The peritoneal pouch is freed and the hernial sac closed, as the hernial sac or peritoneal sac in an inguinal hernia. Having done that, the sacrouterine ligaments are exposed, and it is possible by shortening them and bringing the edges of the fascia together to draw up the rectum and restore some of the attachments of the rectum at the sides of the sacrouterine ligaments, and to get fascia very much as Dr. Watkins has described, although approaching the problem from a little different direction. Where you have protrusion of the anterior wall of the rectum, the fascia must be brought up and drawn upward, and you have more of a transverse slit, as described by Dr. Watkins. The removal of the peritoneal pouch, I think, is a great help in curing these cases of high rectocele in which the pouch is elongated.

DR. GEORGE H. NOBLE, ATLANTA, GEORGIA.—There are two general conditions in cystocele requiring attention, one due to trauma, and the other to excessive deposits of loose connective tissue.

Those due to trauma may be divided into two subdivisions: the first due to lesions of the fascia underlying the bladder, and the other to lesions of the uterine supports and pelvic floor. The cases due to excessive deposit of loose connective tissue may be divided into two subdivisions, also: first, deposits of loose connective tissue around the uterus and below the base of the broad ligaments; second, cases including the above with deposits of loose connective tissue in the middle portion of the cervix uteri.

The general principles involved in operating are constant, though the technic may vary according to the divisions mentioned or the operator's ideals.

Fascia lying beneath the bladder should be brought together from all directions to distribute tension on all sides of the pouch between uterus, broad ligaments and anterolateral walls of the pelvis. For this purpose, the kite or diamond shape excision of anterior vaginal wall (including fascia) is eminently satisfactory.

Excessive deposits of loose connective tissue are usually connected with pronounced uterine descensus, or hypertrophic elongation of the middle portion of the cervix, with eversion of the vagina. Such cases require either hysterectomy or resection of the portio media with wide dissection, the latter for the purpose of replacing loose connective tissue with scar tissue.

In all cases, lesions of the uterine supports and pelvic floor should be repaired.

I deal with large rectocele differently from most people. In the first place, after separating the rectum from the vagina, I evert it through the anus, cut off the sac, and close the hernial opening by bringing the normal rectovaginal septum to the fascia, covering the inner layer of the levator muscle, suturing with kangaroo tendon; then close the perineum in layers. Small rectocele is dealt with as in ordinary perineorrhaphy, by passing suture well around the hernial opening in the rectal fascia, and bringing the upper edge of the hernial opening down to the fascia covering the inner surface of the levator ani muscle.

DR. WATKINS (closing).—I thank Dr. Holden for his kind remarks, which, I am sure, are more complimentary than deserved. We have attempted in case of high rectocele to bridge over the lesion by means of the levator ani and the broad ligaments, as Dr. Danforth has suggested, but the results were very unsatis-

factory. We approve of obliteration of the deep culdesac, which Dr. Studdiford mentions and which is noted in the paper. We have become thoroughly convinced that wide dissections in cystocele are unnecessary and often harmful, for reasons given in the text. It is never necessary to narrow the vagina except as it is narrowed from excision of redundant mucosa. I have been familiar with Dr. Noble's operation for rectocele since he described it, many years ago. I have not had the courage to do it, although otherwise it has had my approval.

DR. OSKAR FRANKL, Vienna, Austria, read a paper entitled **Relation between Placenta and the Secretion of Milk.** (For original article, see p. 399.)

DISCUSSION

DR. HUGO EHRENFEST, ST. LOUIS, MISSOURI.—The interrelationship between the breast and genital tract has, of course, been known for a long time. The gross stimulus must come from the genital tract. We know that breast development coincides with the menarche; that during menstruation often some stimulus reaches the breast; that at the time of the menopause there occurs an involution of the breast glands. As we now understand the problem of menstruation, we might assume a relation of breast changes more directly to corpus luteum function. That gives us some clue as to what probably happens during pregnancy. We know that during pregnancy ovulation ceases; the corpus luteum becomes larger and persists. We might then assume that it is the large corpus luteum which causes the growth development of the breast in the course of pregnancy. The more difficult problem, however, is to explain why the lactation process, the real functional activity of the hypertrophied breast gland, does not begin until labor is over. Professor Frankl deals with this problem. Naturally, outside of the ovaries we have to consider in this connection the uterus and its contents, the uterine wall, the fetus and the placenta. He has referred to the manner in which these various factors have been excluded. It is a common observation that a Porro extirpation of the uterus, does not interfere with lactation. This proves that involution of the puerperal uterus, as has been claimed, is not the deciding factor in starting lactation. Observations in cases of hydatidiform mole exclude the fetus as the activating factor. Thus, by exclusion, the placenta suggested itself as the determining factor. The placenta, however, was not acknowledged to be an organ endowed with internal secretion. Some of the experiments made with placental extracts were suggestive, but never conclusive, because after all, one always had to discount the possibly misleading effects from the injection of protein substances, i. e., possibly nonspecific effects.

In experimental endocrinology most reliance always is placed on the results of the successful transplantation of organs which are functionating abnormally. Just in this manner Professor Frankl has solved the problem of the inner secretory function of the placenta and its relation to lactation by his ingenious experiments, which he described in the paper just presented.

There is another interesting side to this subject. Experiments made by Herrman demonstrated that a lipoid isolated by him from the corpus luteum biologically and chemically is identical with a lipoid extracted in the same manner from fresh placenta. The most characteristic effect he obtained with these very powerful lipoid extracts was a growth stimulation of the breast gland even in new born animals.

Still another very important conclusion can then be drawn from Professor

Frankl's experiments. If corpus luteum and placental lipoids are identical, we can readily support Professor Frankl in his assumption that during pregnancy the placenta takes over or reinforces the activity of the corpus luteum. I consider this a point of great significance in relation to certain therapeutic suggestions that have been made, and which in our literature do seem to play an important rôle. If the placenta supplants or reinforces corpus luteum function, it is difficult to see how corpus luteum injections could ever be required in early pregnancy to overcome certain toxemic symptoms, just at the time when the chorion epithelium surely is most active.

We have read in American and also in German literature about the wonderful effect of placental tissue and extracts in increasing the milk supply of the nursing mother. It is hard to understand any such effect of placental extract when we are in possession of scientific proof that the placenta stimulates growth of the breast glands, but undoubtedly also actually inhibits the production of milk.

Having been familiar with Professor Frankl's experiments for some time, the thought had occurred also to me that there exists a striking analogy between the appearance of the menstrual flow coincident with retrogressive processes in the corpus luteum, and the appearance of milk after the elimination of the placenta, both phenomena respresenting a dissimulation process setting in when the distinctly assimilating effect of corpus luteum or placenta has ceased.

I express to Professor Frankl my deep gratitude for having brought this interesting subject before the Society, and I ask him to consider in his concluding remarks the practical aspect of his experiments in regard to the use of corpus luteum extract in the treatment of the toxemias of pregnancy, and of placental extract for the purpose of increasing the milk supply of the nursing mother.

DR. RUDOLPH W. HOLMES, CHICAGO, ILL.—Why is it that most of the animals eat their placenta if it is not a part of the process of lactation?

DR. ARTHUR H. CURTIS, CHICAGO, ILL.—I would like to ask Professor Frankl whether he has given these pregnant mice any other subcutaneous implantation of foreign proteid material and has noted the effect of it on the offspring. Also, whether he has implanted other foreign material subcutaneously, noting whether or not there was a toxic effect, or whether possibly transplantation of the placenta may not be of this nature rather than showing a specific action.

DR. FREDERICK J. TAUSSIG, ST. LOUIS, MISSOURI.—I would like to ask Dr. Frankl how long the placental implantations lived, or how long they may continue to function.

DR. CAREY CULBERTSON, CHICAGO, ILL.—I should like to ask Dr. Frankl what part of the placenta he used, whether it was the trophoblast, that is, early chorionic cells, or placenta at term. There is a distinct difference between the two. After midpregnancy there is a marked disappearance of the trophoblast, which is active very early during the development of the chorionic villus, whereas at full term the placenta is converted into old connective tissue, with a relative or marked degeneration of the trophoblasts. I ask this question for the reason that before the placental extract was put on the market I was provided with it by one of the manufacturing chemists, for the purpose of using it clinically in obstetrical wards in determining whether it was of any value or not in increasing the secretion of milk. I reported to the firm it was of little or no value and had no influence whatever in modifying in any way the secretion of milk in the mother. There were then later reports, as Dr. Ehrenfest has indicated, to the effect that the secretion of milk has markedly increased by feeding

the woman placental extract. At the time I tried to secure from the manufacturers through their agents some statement as to what part of the placenta had been used, whether they used early chorionic villi, early placenta, or placenta at full term to make the extract, and I was unable to get an answer to that question.

DR. HENRY T. BYFORD, CHICAGO, ILL.—To try to find an internal secretion or a specific stimulant in the placenta responsible for the secretion of milk seems to be complicating a subject that in the last analysis will probably be found to be quite simple. Instead of being a producing organ like the ovary and endocrine glands, the placenta is an organ that is produced during pregnancy for a specific function. Whatever action it may have upon the secretion milk is probably a nonspecific protein stimulant and entirely secondary. The genital influence connected with pregnancy is undoubtedly due to the functions of the ovaries aided by the various endocrine glands. The mammae are stimulated by this influence which, when the uterus is evacuated and reduced to a comparatively quiescent state is suddenly concentrated upon the mammae, while the placenta, having been expelled, must stop exerting any influence from one to three days before milk is secreted, and when its influence should be needed most.

There are many nonspecific stimulants to the secretion of milk, such as nursing, hot nourishing drinks, and perhaps certain proteid or other stimulants acting by way of the blood or nervous system, and it is right that the placenta should be experimented with; but too much time is apt to be consumed in premature discussions.

DR. FRANKL (closing).—As to the question of the age of the placenta which I implanted, I will say that I worked on animals about ten to twenty days before expected delivery and transplanted on them placentae of mice in the same stage of pregnancy. This, however, is not very important for experiments on rodents, because in them the trophoblast persists until the end of pregnancy. There is no degeneration of the trophoblast as occurs in human placenta.

The question was asked whether I tried the transplantation of the ovary in these experiments. No, I tried to replace only a missing organ for the purpose of my studies. The grafted placenta remains active about four weeks, as a rule. After the fifth week it disappears.

I am not convinced that the injection of placental extract preparations or placental tissue increases the secretion of milk. If there was a simple way of treating insufficient lactation, it would be practiced in all clinics of the world. We had no results from using placental extracts for this purpose. With lactation already established every factor which produces hyperemia should theoretically increase secretion, but we have no evidence that this occurs with injection of placental extracts.

DR. HAROLD C. BAILEY AND DR. WILLIAM P. HEALY, New York City, read a paper entitled **Follow-up Results of 908 Cases of Uterine Cancer Treated by Radium.** (For original article, see p. 402.)

DISCUSSION

DR. WILLIAM S. STONE, NEW YORK CITY.—I am naturally much interested in this report by Dr. Bailey and Dr. Healy because the majority of cases I have examined with them and have discussed frequently the treatment of the cases. I think perhaps I ought to explain a little my personal attitude.

Last night I was introduced as the radium fiend of New York. I assure you,

that is not my reputation in New York. I flatter myself that most men think I proceed very cautiously in this work. My work is really that of a policeman and I am a good deal of a pessimist. I have adopted my present attitude in this matter during the past two years. In all this list of cases the percentage of cures cannot be compared with anything in regard to surgery, because, with few exceptions, it represents a definite percentage of cures in cases in which surgery can do nothing at all. In some of the cases that Dr. Bailey has classified as operable, I disagree with him a little bit about the stage of the disease. I am rather inclined to think that they were a little farther along in the disease than he thinks, and the term operability has such a wide variability and is so dependent upon the personal equation, that it is a difficult term to use in any scientific way. In addition to that, I have nine private cases that were seen so early that I thought a simple hysterectomy could cure. These were cases of cancer of the cervix. Of these nine cases, three have passed the five year period, and the last one has gone a year and a half since the termination of treatment. All of these cases are well at the present time. As I say, three of them have passed the five year period. These cases I deliberately radiated, recognizing that surgery probably could have cured them also.

So far as cancer of the cervix is concerned, I believe now we have got into another era in the discussion of the subject, and still most of us believe in radium as a palliative measure. I think that we must in the light of these statistics consider the thing somewhat from a curative standpoint, and I think in individual instances it may often depend on the resources that the patient has, whether she has the benefit of the best surgeon, or whether she has the benefit of a very experienced man in the use of radium.

As far as cancer of the body of the uterus is concerned, while I believe we have shown radium has been curative, at the present time in the presumably early cases I cannot conscientiously rely upon radium alone, for the reason that I cannot see the lesion, I do not know exactly where it is, and I do not think we are quite ready, if we have the surgical resources, to treat the case with radium alone.

In regard to palliation, all has not been said that should be said. There are certain classes of cases in the terminal period of the disease that have been made much worse. It is awfully hard to estimate how much palliation you give in any case, and by how much life has been prolonged, but I am sure that there is another side to the palliative treatment. Undoubtedly some of our cases have been made worse, they have lived a shorter time, and have suffered more.

In the terminal period there are two kinds of cases, one in which the local disease is far advanced, and the general condition shows they are in the terminal period. These cases should not be treated. The other class is pretty close to the terminal period, and the woman's general condition appears to be good on examination, but you will find the case is very likely advanced, and the nutrition of the tumor is beginning to be disturbed. It has not been disturbed before that. There is no toxemia, and so on, but you can see it is already breaking down. If you use radium in that case you will make the patient prematurely worse. These are the hardest cases to withstand.

With reference to the technic, I am not so enthusiastic about external radiation with the x-ray. I believe in advanced cases as a palliative measure it should be done, but there is one thing I have learned from observation of the effect of these agents upon tissues in other parts of the body, that it is quite necessary to observe the nutrition of the normal parts surrounding the tumor. I am not quite sure but what we are going to have a backfire from the intensive use of the x-ray. It is dangerous, and the dangers may not be shown sometimes for

several months, and the x-ray does cause a change in the nutrition of the normal surrounding parts, which is a great disadvantage.

DR. OSKAR FRANKL, VIENNA, AUSTRIA (by invitation).—I agree with very much Dr. Bailey has said.

When we began radiation of these cases in our clinic we started with radium. The primary results were excellent, but after a short time they proved disappointing. Later, when we learned to improve the technic of radium application, the primary results became much better, and yet the ultimate results were absolutely unsatisfactory. It was only when we began to combine radium with the x-rays that our results were markedly improved.

Regarding the operative treatment of these cancerous cases, we should operate whenever we think it is possible to do so to advantage. After operation we treat these cases by radiation, but only by means of x-ray, excluding radium. Since we have followed this method we have cured very many cases. I have not the statistics at my disposal at this time.

I am sure Dr. Bailey has also studied the interrelationship between mature carcinoma, and the finer histological structures of the growth, and the results of radium and x-ray treatment. In our clinic, as in other clinics, it was found that there is a very great difference between radiation of the one or another carcinoma, according to its maturity. We have carcinomas which will melt like butter in the sun, and again others which prove very refractory. We have patients who respond very satisfactorily, but these are cases of solid carcinoma that have reached an advanced state of maturity. The old surgeons knew that an epithelioma of the skin cannot be compared in malignancy with the carcinoma of the tongue. The worst carcinomas, according to our experience, are always those of the glandular type of low maturity.

DR. GEORGE GELLHORN, ST. LOUIS, MISSOURI.—Limiting myself to cancer of the cervix, I will say that Dr. Bailey's figures confirm the continental statistics according to which radiation rivals, if not excels, the results of surgery by a permanent and absolute cure of between twenty and twenty-five per cent. It must, however, be obvious to you that if the rest of us are ever to achieve such results, larger quantities of radium must be at our disposal than most of us possess. Those of us who have only 100, 130 or even 150 milligrams of radium, are forced to combine radium with operation in early cases. We first destroy the cancer in the cervix by radium, and then remove by operation the tissues outside of the uterus where the radium cannot penetrate. To do this, and this is the point I want to stress, we must resort not to an ordinary hysterectomy, but to the extended Wertheim operation despite its high primary mortality.

The second point I wish to make refers to certain forms of recurrences. Local recurrences in the operative scar in the roof of the vagina are not nearly so frequent as they formerly were when direct contamination was less carefully avoided during operation. We are now more apt to meet with recurrences in the outer portions of the parametria close to the pelvic walls. In this location the cancerous foci are not directly accessible to the radium capsules or needles, and any intravaginal radium application carries with it the danger of vesical or rectal fistulae. For this class of cases the suggestion made by Bumm a year or so ago seems to me worthy of attention. Bumm approaches the pathologic process by the paravaginal route, and I have followed his lead, substituting needles for capsules. It is easy to make a small puncture close to the tuberosity of the ischium and, with two fingers in vagina and rectum, guide a prostatic needle containing radium into the tumor itself. Two or more such needles can be introduced and left in place to correspond to 300 or 400 mghr. I have done this three times thus far.

In the first case the radium dosage was insufficient, and the tumor seemed to have been stimulated to faster growth. In the second case, the tumor disappeared promptly, but it is too soon to speak of definite results. The third case is altogether too recent; moreover, deep x-ray treatment was added to the radium application. I wonder whether others have had experience with successful treatment of such deposits far out to the side of the pelvis.

DR. JAMES E. KING, BUFFALO, NEW YORK.—The thing which most impressed me during the reading of the paper is the undertone of disappointment that pervades it. It suggests what many of us feel, that radium is not fulfilling the high hopes at first entertained as to its value as a cure of cancer of the cervix. These reports, too, have great significance for those of us who have only a limited amount of radium at our disposal. I have had 100 mg. of radium for about a year and a half and, it seems to me, that it is time for us to learn whether the use of these small quantities is of as great value as the larger amount.

In estimating the value of radium as a cure for cancer we often ignore a fact which we all learned in the operative treatment of cancer. We all realized that there was some factor other than the technic and skill of the surgeon which determined whether an operation was to be successful as a permanent cure, and upon which also depended the time at which recurrence would take place. For this reason none of us, as far as the surgery of cancer was concerned, ever had the temerity to make a prognosis as to cure, or to predict the time of recurrence. This factor we know nothing about. Call it immunity, resistance, or what you will, but until we do know what that factor is we cannot determine the practical value and the dose of radium for a given case. On account of this unknown factor radium as a cure for cancer of the cervix shares the same uncertainty as its treatment by surgery.

I would be glad to have Dr. Bailey express his opinion as to the smaller amounts of radium, compared with the amounts available in his own and other institutions.

DR. WILLIAM P. HEALY, NEW YORK CITY.—In 1921, when Dr. Bailey went to the Department of Obstetrics at Cornell, and I was asked to succeed him at the Memorial Hospital and to take over the work in gynecology, I was sure I had a fairly good knowledge of cancer of the cervix because I had been associated with Dr. Howard C. Taylor at the Roosevelt for twelve years, and also had my own service at Fordham Hospital. Yet when I went to the Memorial Hospital I discovered very promptly we had been greatly restricted on those services in our efforts to take care of cancer of the cervix. I found that at the Memorial Hospital we were taking care of cases I had never before appreciated could have the lesion treated to advantage by any physical agent.

In 1921 there were eighty-five primary advanced cases of cancer of the cervix totally beyond the possibility of surgery, and of those cases there are thirty-one alive today, and twelve absolutely free from any evidence of cancer.

Last Wednesday, a week ago, there came to my clinic a woman thirty-five years of age, who applied for treatment in July, 1921. She was a graduate nurse, yet when she applied for treatment the disease had completely invaded the cervix, so that it was like a horse collar with a solid ring, with the base of each broad ligament solid, and the uterus firmly fixed. She came in a week ago today with no local evidence of disease in her pelvis, apparently in perfect health and with no symptoms of any kind. When we asked her to return in September for the next examination, she asked that it be put over until October because she was going to attend a convention some place in the western section of the country.

She is one of the twelve cases entirely free from disease. These are the primary advanced cases.

DR. BAILEY (closing).—I was much interested in what Professor Frankl said in regard to the maturity of the cells. We have based our work upon the morphology of the cell, but in a somewhat different way. The squamous cell carcinoma is a resistant tumor, but, on the other hand, it is a tumor which tends to be very sharply localized and therefore, our results are better from that particular type than from any other. The columnar cell carcinoma, or the one which appears like adenocarcinoma, is easily affected by radium. The trouble is that it may extend far beyond any point where one may judge it to be. It is a rapidly spreading tumor and goes outside the bounds where you can give a satisfactory dosage. A third kind is the basal cell tumor, which is easily affected, but is not common in this location.

In regard to Dr. Gellhorn's question, I believe that a man with a small amount of radium has no business attempting to treat these operable cases with radium alone. It should be used in conjunction with operation. I feel certain on that point. In regard to recurrences in those areas that are far out in the pelvis, the paravaginal incision suggested by Dr. Gellhorn is interesting.

There is another point I wish to speak of, because it is so seldom referred to, and Dr. King brought it out. There is no way of telling what the reaction is going to be. Individuals have a different type of immunity against cancer and the cells have a different virility, and besides that, there is a general blood reaction to the irradiation in some cases that does not occur in others. There is a limiting of the tumor growth by a dense infiltration of plasma cells and leukocytes.

DR. CURTIS.—How about the treatment of carcinoma of the fundus?

DR. BAILEY.—Carcinoma of the fundus should be operated on in conjunction with the use of radium. It should have, first of all, a heavy sterilizing dose of radium, and then the uterus should be removed.

DR. FRED J. TAUSSIG, St. Louis, Mo., read a paper entitled **Contributions to the Pathology of Vulvar Disease.** (For original article, see p. 407.)

DISCUSSION

DR. OSKAR FRANKL, VIENNA, AUSTRIA (by invitation).—I wish to add only a few words to the excellent paper of Dr. Taussig. We must feel very grateful to him for having pointed out the fact that kraurosis is not a disease, but only a symptom. It does not offer a sharply defined pathologic picture, and that is the reason why I contradicted Veit who took the stand that kraurosis vulvae is a place in which a carcinoma will develop. We must not make the mistake and confound kraurosis vulvae with leukoplakia, and I think Dr. Taussig is right, when he says that carcinoma originates on the basis of a leukoplakia.

I cannot agree with the essayist as to the word "precancerous." I think we would better eliminate the word precancerous or precarcinomatous from our medical terminology. There does not exist in my judgment a precarcinomatous condition. We either have a carcinoma or no carcinoma. Precarcinomatous is a word without a real meaning. We can say that the leukoplakia vulvae is the place of predilection for the development of cancer. The word precarcinomatous unavoidably has led to many operations that are not indicated. I believe we should drop this term from our medical terminology.

DR. WILLIAM P. HEALY, NEW YORK CITY.—At the Memorial Hospital, New York City, we see a fair number of cases of leukoplakia of the vulva, and we have treated some of them with electrocoagulation, with reasonably good results so far as clearing up the lesions is concerned. This method of treatment has only been used for a short time on several of these cases, so that we do not know how permanent the result is going to be, but the immediate result has been the replacing of the leukoplakic area with normal skin subsequently.

As a part of the contribution to the pathology of vulvar diseases, I want to draw attention briefly to a case of estheomene under my supervision which I published early last fall in the AMERICAN JOURNAL OF OBSTETRICS AND GYNECOLOGY. It was an unusual case in which we finally concluded that the etiologic agent in maintaining the lesion was evidently a proteus organism, because we finally cured this young woman by the use of proteus vaccine.

DR. TAUSSIG (closing).—With reference to the use of the word precancerous, I spoke of "so-called" precancerous lesions. We should have some term for those lesions, favorable soil, on which cancer tends to develop, and if some one can suggest a better name for these lesions I would be glad to use it. Leukoplakic vulvitis is followed in fifty per cent of the cases by cancer. This soil predisposes to cancer and some term should be adopted to designate that kind of lesion.

As to the treatment of these cases, I feel anything in the way of cauterization or radium produces but temporary effect. I have tried radium in a number of cases with only a short period of relief. The x-ray likewise gives temporary results. Excision, on the other hand, gives satisfactory results. The percentage of recurrences after excision would not run over twenty per cent. The entire figure-of-eight involvement around the vaginal orifice and around the anus, with the perianal tissue as well as the perivulvar must be removed in order to prevent such recurrences.

DR. JOHN OSBORN POLAK, Brooklyn, N. Y., read a paper entitled **Further Study of the Retained and Adherent Placenta**, an abstract of which follows:

Placenta accreta is a pathological entity and must not be confused with retention or adhesion of the placenta. In accreta the placenta is directly attached to the underlying muscle structures—no serotina can be demonstrated, neither can any line of cleavage be found. The placental tissue erodes itself into the underlying muscle and makes one continuous mass with the uterine wall. Such cases are of extreme rarity; only one instance of placenta accreta was observed in the study of six thousand deliveries in the obstetric service at the Long Island College Hospital. Three other cases have been seen by the writer in thirty years of consultation practice; of these only one recovered, the others died of hemorrhage, sepsis and perforation of the uterine wall which resulted from attempts at manual removal.

In placenta accreta, as there is no serotina, there is consequently no line of cleavage, hence when manual removal is attempted the placenta is removed piecemeal and with it portions of the uterine wall. This opens the uterine sinuses and subjects the patient to the dangers of excessive blood loss, perforation and sepsis. Its etiology, according to Frankl and confirmed by histological studies in our cases, is due to conditions which produce an absence of the endometrium. These are previous manual removal of the placenta, repeated curettage, uterine scars, fibromyomata and atrophy of the endometrium. Three of our cases had

had previous manual removals and one had been curetted three times just prior to becoming pregnant. Absence or atrophy of the endometrium permits the ovum to implant itself upon a basal membrane and erode itself into the muscular wall which is unprotected against the erosive action of the villi by the absence of a well defined decidua. The result is a truly adherent placenta invading, and intimately attached to, the uterine muscle with no line of cleavage.

The symptom complex is a placenta which fails to separate with the classical signs of separation: i.e., rise of the fundus, descent of the cord and vaginal bleeding. The uterus does not bleed in placenta accreta because the placental site remains completely covered with the attached placenta and no sinuses are torn open. Retraction occurs, except at the placental site, hence the shape of the uterus is usually asymetrical, one cornu being more prominent than the other. Adhesion may be presumed if the placenta is retained for over two hours after the delivery of the fetus, in the absence of the classical signs of separation.

Under such conditions, having conducted the placental stage on proper lines: i.e., avoiding fundal manipulation and attempts at forceful expression by Credé, in the absence of the signs of separation the writer suggests that under most careful surgical asepsis, the uterus be explored with the sterile gloved hand, and if no line of cleavage can be demonstrated, attempts at manual removal be discontinued.

Two plans of treatment may now be instituted: (1) After aseptic preparation of the approaches, the cord may be tied and cut flush with the cervix and the interior of the uterus firmly packed with washed iodoform gauze. This will occasionally stimulate contractions and if the adhesion is incomplete cause separation in the course of forty-eight hours. If the placenta does not separate and deliver itself on the withdrawal of the gauze, further intravaginal procedures should be discontinued. (2) Abdominal hysterotomy offers a clean field for attempts at separation, and if no line of cleavage can be demonstrated on uterine incision, hysterectomy should be the method of choice.

DISCUSSION

DR. BARTON COOKE HIRST, PHILADELPHIA.—There are two points, it seems to me, that might be discussed in Dr. Polak's interesting and instructive paper. First, the incidence of placenta accreta. In upwards of 40,000 cases seen in my private and hospital service, I have only seen one case of true placenta accreta, so that it might be less frequently encountered than Dr. Polak's statistics would lead us to suppose.

The other point is with reference to the necessity of doing hysterectomy. My experience in the single case we have had would indicate that it is not always absolutely necessary. It took four or five days to get rid of the fragments of placenta, but eventually it was accomplished, and the patient ultimately made a perfect recovery.

I recall a case like the picture Dr. Polak showed of fibroid tumor of the uterus complicating pregnancy. When I saw the woman some four or five days after delivery, the separation and removal of the placenta had been accomplished, but there was then infection and necrosis of the tumor and I was obliged to resort to hysterectomy. The patient recovered. I should hesitate to subscribe unconditionally to the doctrine that every case of placenta accreta should be treated by hysterectomy. No doubt it is the treatment giving the best average results in the hands of a specialist, but it might be unfortunate to have the profession get the impression that a case of adherent placenta, difficult to extract, should be dealt with by a major operation.

DR. WILLIAM E. STUDDIFORD, NEW YORK CITY.—In reviewing the cases that have been under my charge at the Sloane in the past four years, we have had no case of this kind, barring one that possibly might be considered in this class, a case in which I feel there is very little to be gained so far as the management of it was concerned. The patient was a woman who had been subjected to a series of curettages, I think four or five, in the preceding five or six years, and who was admitted to the hospital at about the sixth month of her pregnancy with a premature labor, and was seen only by one of the internes. A macerated fetus was delivered, and the woman was sent back to the ward with no note as to the condition of the placenta. She was in the ward for about ten days, with very little bleeding from the uterus, and there was nothing abnormal as far as temperature was concerned. She was discharged at her own request, and reported at the clinic some six weeks later with hemorrhage. She said she had more or less constant dribbling of blood, and in reviewing the history of the case the question of malignancy of the body of the uterus came up. The uterus was enlarged. Under an anesthetic an exploratory curettage was made, and a mass of tissue removed which to the eye appeared to be malignant. An immediate hysterectomy was done, and to our surprise we found, instead of malignancy, placenta with the usual granulation tissue of the wall of the uterus. She had carried it for a period of six weeks, with no temperature, no infection, and the placenta had invaded the uterine wall. I can readily see that a case of this kind by manual manipulation, unless it was done under the most careful precautions, would lead to a very high mortality following sepsis, or any attempt to remove all the particles of placenta would cause perforation of the uterus.

I feel the condition must be one of extreme rarity, and as to the question of hysterectomy I quite agree with Dr. Hirst that it would depend a good deal on conditions under which the cases were managed. After attempts at manipulation had been made under unfavoroble conditions, then the question of surgical treatment or a radical operation would be the only thing to consider. On the other hand, if the case is carefully managed from the start, it is quite possible the uterus might be preserved.

The question of etiology, to me, is the one of repeated curettage. It reverts back to the old subject which was discussed many times, the objection to curetting every uterus for slight bleeding. Practitioners will curette and curette these cases so thoroughly that the endometrium is practically destroyed, and the uterus is no longer a suitable organ for future pregnancy.

DR. OSKAR FRANKL, VIENNA, AUSTRIA (by invitation).—For a long time I have been greatly interested in the subject which Dr. Polak has brought before us. I distinguish very sharply between pseudoplacenta accreta and real accreta. What we call placental adhesion has nothing to do with placenta accreta. I am absolutely sure that if a placenta five or six or seven days after delivery separates by manipulation, it was not an accreta. When we have a real placenta accreta, its separation is absolutely impossible. In the immense material which passes every year under my eyes I recall but six cases of true placenta accreta, in which the organ was united with the uterine wall without the interposition of a decidua. I have here one slide which corresponds to the picture shown in my book on Gynecologic Pathology published in 1914. The villi are so intimately united with the uterine muscle that you can follow them for half a centimeter underneath the inner surface of the uterine wall.

How can we detach this placenta? Twice I have made postmortem examination on such cases and had the specimens before me on the table. It seemed that one should be able to force the placenta from the uterine wall, but it was

absolutely impossible. I found that I was tearing out pieces of the uterine wall. I know of a case, which occurred outside of our clinic, where a doctor tried to separate such a placenta and in so doing pulled off part of the uterine muscle. Dr. Polak correctly spoke of an anatomical placenta accreta in which a decidua does not exist.

With regard to the etiology, repeated curettage plays an important rôle, and so does submucous myoma. I saw cases of placenta accreta after a severe endometritis, but such cases are very rare, because with an endometritis of this sort pregnancy is not probable. In one case which I studied the entire mucous membrane was destroyed and the ovum was implanted directly in the uterine wall.

In a case of septate uterus, sometimes the implantation of the ovum on the septum leads to placenta accreta, because the septum often has an underdeveloped mucous membrane without glands, therefore no decidua forms. Where the decidua is absent, separation of the placenta is impossible. I have reported similar occurrences in instances of uterus bicornis, leading to placenta accreta when the mucous membrane of one horn was underdeveloped.

In the case of a total placenta accreta bleeding is impossible. I have seen five such cases. As a rule, in nine cases out of ten, the placenta is implanted without any decidua. In one case I recall the placenta was at least partially implanted on a decidua. I am able to show you the slide of such a case. It is from a myomatous uterus. The patient had a submucous myoma recognized before pregnancy. We advised operation, but she refused. She became pregnant, and was first seen in the third month of pregnancy. I again urged operation because we were convinced she had a submucous myoma; she finally consented. With the sole exception of submucous myomas we are conservative in the treatment of cases of myoma complicating pregnancy. You can see in this slide that she would have had a placenta accreta. We have seen in the clinic three or four such cases. I thoroughly agree with Dr. Polak that in cases of anatomical placenta accreta the indication is definite for extirpation of the uterus. In this slide you see that almost the entire placenta is directly attached to the uterine wall without decidua; only one part, about two centimeters, showing the interposition of a decidua between placenta and uterine muscle.

DR. CAREY CULBERTSON, CHICAGO, ILL.—Dr. Polak has made the point that placenta accreta is a pathological entity, and this may be accepted without much question. Poor decidual formation is a factor in its development, in all probability. In his particular case the presence of a submucous fibroid explains why the mucosa was atrophic, and hence why the decidua had poor material out of which to develop. We should not, however, look for placenta accreta in pregnancy at term only, but also in early pregnancy. I am satisfied that it is a factor in some abortions.

I have two uteri, both of which were removed years ago for uterine hemorrhage, before radium became the accepted treatment for metrorrhagia. I removed one of these organs myself, and the other was removed by a friend of mine, and in neither case had pregnancy been suspected. Chorionic villi and cells of the trophoblast were found penetrating into the muscularis. The question comes up whether in such cases, in which the diagnosis is properly abortion due to placenta accreta, the treatment should be hysterectomy instead of simple curettement; and further, whether such a case would be suitable for treatment by radium rather than by hysterectomy or curettement.

DR. HENRY T. BYFORD, CHICAGO, ILL.—I would like to ask if in these cases there is any immediate danger of hemorrhage or of sepsis if nothing is done. I wonder whether it can be determined immediately, and whether in such a case

it is safe to wait a day or two, make another trial and see whether it is really such a case, before resorting to the extreme measure of hysterectomy.

DR. EDWARD A. SCHUMANN, PHILADELPHIA, PENNSYLVANIA.—I should like to ask Dr. Polak whether he has any information which would lead him to believe that if a placenta accreta were allowed to remain *in situ* indefinitely, it would separate by necrosis, followed by absorption.

DR. FRED L. ADAIR, MINNEAPOLIS, MINN.—I would like to ask Dr. Polak how he differentiates clinically between placenta accreta and certain types of placenta which cannot be accreta, but which have to be removed piecemeal.

DR. POLAK (closing).—In answer to Dr. Schumann's question, I will say that there are photographs in the exhibit of a case from our clinic, operated on by my associate Dr. Jewett, of an abdominal pregnancy with the placenta implanted upon the peritoneal surface of the rectum and posterior uterine wall. This case illustrates the manner in which a placenta becomes attached to the underlying structures in the absence of a protective decidua. This patient was operated upon over a year ago, and because of diffuse attachment, and the absence of any line of cleavage, it was left *in situ*. Dr. Jewett and I examined this woman two or three days ago and the placenta can be found as an insensitive mass in the culdesac, about one-third its original size, still attached to the rectum and the uterus. It is being aseptically absorbed. The same thing might obtain in a uterus, were its interior not subjected to the migration of bacteria, but aseptic absorption and organization cannot take place in the presence of saprophytic organisms.

In reply to Dr. Byford's question, I would say that we have left the placenta in the uterus as long as five days in order to have it separate, and have seen such a placenta delivered spontaneously. What we have tried to bring out in this paper is, that placenta accreta is a different pathological entity from adhesive placenta. In accreta the placental tissue invades and destroys the uterine muscle wall. If you will look at the sections, you will note that the placental villi have grown in between the fibers of uterine muscle, and have penetrated almost to the peritoneal covering. One section, directly opposite the implantation of the cord shows the peritoneum as the only part of the uterine wall that is left intact. If that is so, there is nothing that can be done except hysterectomy, for attempts at separation mean perforation of the uterus. Whether our figure of incidence is wrong or not, I do not know. We simply reviewed the cases five years back and picked out the manual removals. Of these there were eight, and from among this number there was one case of placenta accreta.

The main point, it seems to me, between Dr. Hirst and Dr. Studdiford and myself is that we are talking about different pathologic conditions. Adhesive placenta is not a placenta accreta. Dr. Studdiford's case is illustrative of the latter condition.

Dr. Adair asked how to differentiate placenta accreta and other types of placenta which have to be removed in pieces. I do not know. What we have tried to do, and what we suggest is, that if the placenta is retained for more than two hours without bleeding then we explore the interior of the uterus, and if there is no line of demonstrable cleavage, get out. We can wait if we want to for the placenta to separate, but how long to wait I do not know. I have waited five days. If we cannot get it out without pulling it off and pulling pieces of the uterine wall with it, it is better to remove the uterus. I do not know how to make the differentiation clinically, but I would like some of you to tell me.

Department of Reviews and Abstracts

CONDUCTED BY HUGO EHRENFEST, M.D., ASSOCIATE EDITOR

Collective Review

Some Forgotten Nativities

BY ALFRED ELA, BOSTON, MASS.

THE kinship of obstetrics to religion is much closer than is generally recognized, and is manifested in most curious ways,[5, 7, 8, 9, 10, 11, 24] although in recent centuries frequently concealed. A cross-section of Christianity, including the first century to the twentieth, will reveal much not patent to the superficial observer, a few instances of which are as follows:

I. The earliest extant hymnbook[14] of the Church was lately discovered, and the publication of this once almost canonical book has excited the keenest interest in theological circles, especially in Roman Catholic ones, as to its nineteenth Ode, with its "impossibly early" details of the Virgin birth. A certain "retranslation into the original Hebrew" is a monument of misdirected learning, there being little doubt that the Odes' language was Syriac, and that they were written at Antioch about 90 A.D. This most important Ode (the nineteenth) is still a puzzle, and my attempted solution[9] is still in manuscript for reasons stated in postscript hereto. The upshot of my manuscript (so far as pertinent) is that the Father actively assisted (as foreshadowed in the Obstetrical[9, 32] Psalms of David) the painless delivery by the Virgin of a giant Child, at its own volition and after a protracted pregnancy,[11] then universally believed to be a concomitant of divine paternity.

The possible theologic connection between North Syria and Egypt is a matter of keen controversy,[18] and is here noted merely as perhaps a bead on the chain running through this article.

II. Jesus' prolonged gestation was also believed in by the Sabaean Christians of Arabia (Felix[19]). This southern Arabia has been so sheltered from outside influences that its synagogues today, alone in the Jewish world, are said to use the Targums[15]—commentary in the former vernacular, upon the Scriptures. This South Arabian Christianity is known to very few historians. Some of these Christians were evidently taken by the Arabians of Yemen (the chief local city) during their Moslem invasion of Egypt and of Spain.

III. This invasion of Egypt brought intimate contact with the Christians there, of the Coptic Church, one of the earliest, but whose very existence was forgotten in Western Europe for many centuries. They preserve the oldest extant manuscripts of the New Testament, and constitute one of the most conservative races of mankind. Ac-

cordingly, Christianity doubtless changed them but little from their ancient beliefs,[22, 23] and a detailed comparison of the iconography of Isis[26] with that of the Blessed Virgin would be most interesting and valuable. A single citation[4] of a Nativity on an early fresco in a chapel of the Virgin must here suffice. On a bed (according to a tradition differing from our accepted one, but which has also survived in out-of-the-way Western Christianity) reclines the Blessed Virgin, with the midwife sitting expectant; the Child is not yet in evidence.

IV. Spain was finally reached by the Moslem invaders in two parties centering roughly around Cordova and around Seville.[27] The Moslem rule in Spain has, until recently, been treated from the North, and thus the fundamental differences between the two parties have been overlooked. The Seville section was that of the Arabs from Yemen, who evidently brought many Christians, Sabaeans and Copts. These found congenial fellows among the Spanish Christians around Seville (who apparently had long been under Egyptian influence), and these branches of Christians seem to have assimilated, and to have (under the name of Mozarabs) acquiesced in, and flourished under, the Arab rule. Thus they preserved many rites and doctrines unknown except so far as they have spread to the rest of the Latin Church. As one instance (among many available if my notes were accessible) that sundry phases here presented are not novel, we may refer to a rite still practiced in a political and ecclesiastical colony of Seville.

V. The Canary Islands were in 1344 given over by Pope Clement VI to Don Luis de la Cerda, a grandson of Alfonso I, "with the title of prince, and instructions to conquer and Christianize them."[30] But Don Luis did not go to the Canaries and it was not until the beginning of the fifteenth century that Franciscans from Seville began the evangelization of the Islands.[30] Why and how St. Francis himself was interested in the Nativity we cannot go into here,[6] but we shall proceed at once to the latest mass on Christmas Eve in a great church at Teneriffe, mentioned by a medical scientist[27] and more fully described by him in a work[28] crowned by the French Academy, of which the following excerpt is a literal translation: "Suffice it to say that in one of the principal churches of the island (Teneriffe) on Christmas Day a richly adorned Virgin of wood is placed on the altar. At midnight a priest approaches, opens two doors hinged into the abdomen of the statue, and then, to the great joy of those present, takes out a beautiful pink babe." This rite, now perhaps unique within the Roman obedience, seems to have survived purely because it was on remote islands and thus overlooked in the unifying influence of Roman rule. We shall see that it appears formerly to have been widespread and, at any rate, it fits in with a rite still practiced in the Cathedral city, to wit:

VI. Seville's "Misa del Gallo" has outlived much opposition and excites the wonder of Catholic visitors. Immediately upon the stroke of midnight, as if continuing the action of the Teneriffe rite, the officiating priest takes from the altar an image of the Holy Babe, and sits with it on his knee.[31] This is the identical attitude of Isis with her babe Horus.[25]

VII. Teneriffe's rite may explain what has long been a puzzle to medical antiquarians and others, to wit: the *"vierges ouvrantes"*[1, 2, 3, 16] found in many of the older churches in parts of

France where formerly the cult of Isis was prevalent. These images of the Virgin have hinged doors in the abdomen, which, when opened, disclose within an image of the Child, or even of the Trinity.[17] These are so repugnant to the present feeling of the Church that they generally are opened reluctantly—or even this is made impossible by iron bands rivetted by episcopal order. That the cult of Isis was deeply rooted in Gaul during the early part of our era has been a commonplace of history for years, and has lately received some astonishing support.[13, 20, 21]

VIII. One therefore cannot be surprised at this possible resolution of the age-long riddle left us by Firmicus Maternus (about 350 A.D.), viz.: "In the sacred rites of Isis a log is cut from a pine tree. The central portion of this log is carefully excavated, and within it is buried an image of Osiris made of seeds."[12, 33] Thus we are brought back to the foot of the statue of Isis, and the fit symbol of the serpent with its tail in its mouth.

POSTSCRIPT

The above is but a fragment of what I had in mind when glaucoma overtook me. Remembering but little of my material (which was in notes then before me), I have been forced to rely on what remains from that little. At the outset of my blindness I made a grim joke (later corroborated by curious coincidences), to wit: "I have tried to lift the veil of Isis, but she has forestalled me by casting her veil over me." Accordingly, I must offer the above chain with most of its beads lost and those remaining much dimmed.

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1891, p. 313: "Il me suffira de dire que, dans une des principales eglises de l' ile, (Ténériffe), le jour de Noël, on place sur un autel une Vierge de bois, richement paree. A minuit, un prêtre s'approche, ouvre deux portes ménagées dans le ventre de la statue et en retire, à la grande joice des assistants, un joli bébé rose." (29) *Whishaw, B. and E.:* Arabic Spain, 1912, *passim.* (30) *Whishaw, Mrs. B.:* My Spanish Year, 1914, pp. 213, 214. (31) Ibid, 178-182, esp. 181-182. (32) In this place I can only suggest that our accepted Hebrew text is the result of severe excisions by generations of ancient Hebrew editors, and that this text has in turn been much mollified in our English translation. (33) What is here meant by "pine", "Osiris" and "seeds" we need not here discuss.

Selected Abstracts

Infection of Internal Genitalia

Salomon and Rath: The Origin of the Genital Flora. Part I. The Origin of the Intestinal Bacteria. Zeitschrift für Geburtshilfe und Gynäkologie, 1922, lxxv, 141.

As a preliminary to the study of leucorrhea, the authors determined first to make a study of the origin of the normal vaginal flora in the newborn child. They, therefore, studied simultaneously the first invaders of the vulva, vagina, mouth, rectum and skin, as well as the bacterial content of the birth tract of the mother and of her breasts during labor and the puerperium. In the larger proportion of cases, contents and wall of the rectum were sterile at birth, yet organisms invariably appeared within a few hours, far too soon for a descending invasion from the mouth. Cases delivered by cesarean section showed a later invasion than those delivered by the natural passages, while cases long in labor with ruptured membranes often showed earlier invasion. Of great influence upon the rectal flora were the passage of fecal material by the mother during labor, the bacterial content of the mother's vagina, the clothing and the bath of the newborn, the hands of the attendants, the air and the contents of the room. The regularity with which the first organisms appeared was of importance not for the intestinal flora alone, but also for the vaginal flora, as the authors showed in later papers. MARGARET SCHULZE.

Salomon, R.: The Origin of the Genital Flora. Part 2. The Origin of the Mouth Organisms. Zeitschrift für Geburtshilfe und Gynäkologie, 1922, lxxxv, 306.

The origin of the flora of the child's mouth is in great degree dependent upon the flora of the mother's vagina, upon the organisms on the mother's breasts, in lesser degree upon the bath water, the hands of attendants and of the mother, the clothing, rubber nipples, etc., and finally upon the surrounding air.

Of great importance for the mouth vegetation is the nature and length of the labor, the time of rupture of the membranes, the number of internal examinations, operative interventions, the nature of the puerperium, as well as the state of health of the child.

In the first twenty-four hours, twelve different microorganisms could be cultivated; in the first ten days, twenty-one varieties. The gram positive cocci (micrococci, streptococci, staphylococci and gram positive diplococci) predominate, particularly in the first few days over the gram positive rods (vaginal bacilli and the like). In the following days, the latter increase in number, but never reach the

numbers of the gram positive coccal forms. The relations are here, therefore, just the reverse of those in the rectum. The bacterium coli is not, as is generally accepted, a rare mouth organism in the newborn. Streptococci and staphylococci show all the characteristics of virulence without giving rise to clinical manifestations. Their hemolytic properties are of no significance.

Since the possibilities of an invasion with organisms and infection of the mouth and gastrointestinal tract are so great, the problem of a rational care of the newborn is that of keeping away all possible pathogenic organisms, and thus limiting the bacterial invasion to those which are known to be harmless.

MARGARET SCHULZE.

Salomon, R.: The Origin of the Genital Flora. Part 3. The Origin of the Vulvar Organisms. Zeitschrift für Geburtshilfe und Gynäkologie, 1923, lxxxv, 554.

In most cases, the vulvae of children were found to be free of organisms immediately after birth, but after from five to seven or eight hours, the first organisms appeared to increase progressively in numbers. After ten hours, 28 per cent of all vulvae (compared to 57 per cent of rectum or mouth) were inhabited by organisms, after twenty-four hours 79 per cent of the vulvae as compared with 89 per cent of the rectum, and 95 per cent of the mouth. After the second day, organisms were to be found on all.

In the first twenty-four hours, fourteen; in the course of the next ten days, twenty-three different organisms could be identified. The vulvar flora shows no such constancy as the rectal flora. After the third day, rods and cocci were found in about equal numbers. The type of vulvar organisms is dependent on many circumstances. The bath is of less importance than is generally considered. Children delivered by cesarean section show vulvar organisms later than those delivered by the natural passages, and they differ quantitatively and qualitatively from the latter. On the second day, there appears a "transitional catarrh" of the vulva depending upon bacterial invasion, such as has been described for the rectum.

The type of vulvar bacteria depends upon the rectal and mouth flora of the child, the vaginal organisms of the mother during labor, the type of labor (breech presentations, etc.), operative interference, the time of rupture of the membranes, the number of internal examinations, the condition of the mother during pregnancy, and in the puerperium, the time the child is first put to breast and its condition, as well as the asepsis in its care. A knowledge of the vulvar flora is important for an understanding of the vaginal types, since no organism can be found in the vagina which has not passed the vulva.

MARGARET SCHULZE.

Sternberg: A Contribution to the Nature of the Saprophytes of the Female Genital Canal. Zeitschrift für Geburtshilfe und Gynäkologie, 1921, lxxxiv, 447.

The author's experiments were conducted upon nonpregnant gynecologic patients who did not have leucorrhea or any inflammatory lesion of the lower genital tract. Agglutination tests with the patient's serum were made against the streptococci or staphylococci which were first isolated in pure culture from the patient's secretions. A high agglutination titre was accepted as evidence of potential pathogenicity of an organism, as the body does not develop agglutinins against saprophytes. It was found that vaginal organisms which were not giving rise to clinical symptoms nevertheless might have virulent characteristics, since they formed agglutinins in high titre. In most cases the streptococci developed more antibodies than the staphylococci. It was found incidentally that hemolytic

properties are no indication of the virulence or lack of virulence of a streptococcus. It has long been known that the vagina is self disinfecting. It soon rids itself of foreign bacteria. This ability has been ascribed in part to the acid reaction of the vaginal secretion, in part to leucocytic action, and in part to overgrowth of the foreign organism by the normal vagina flora. The author believes that this self-disinfecting power is of little value in keeping streptococci and staphylococci harmless, but that this is done rather by the general resistance of the body. For instance, as a result of the reduction of the general bodily resistance by an operation for cervical carcinoma, seemingly harmless streptococci frequently lead to peritonitis and death. The author believes, therefore, that the term saprophyte for these vaginal organisms is misleading, since they live harmless as saprophytes only so long as the general bodily resistance balances them, and may quickly become parasites if any factor lowers the protective powers of the organism and these are not developed, or are developed insufficiently or too slowly. MARGARET SCHULZE.

Bigler, W.: The Origin and Treatment of Vaginal Discharge. Schweizerische Medizinische Wochenschrift, 1922, lii, 1193.

The usual vaginal discharge is caused by specific and infectious organisms such as: gonorrheal, septic, tuberculous, diphtheritic. Tumor growth or other disturbances of the inner genitalia, even pregnancy may also cause a discharge. Beside these causes the discharge may be from a nonspecific cause or from other flora not commonly encountered in the vagina. There are three main classes: the vaginal bacillus of Döderlein with a few leucocytes, lymphocytes and squamous epithelium in the discharge; the second type a gram positive diplococcus and vaginal staphylococcus causing a whitish acid discharge; and the third type, a gram positive or negative coccus in short or long chains, sometimes of tetragenous form, different type of sarcinae, pseudodiphtheria and saccharomyces, fusiform bacilli and even trichonomatous type. The discharge is of yellowish color and may be either acid or alkaline.

The treatment consists in attempting to discover the underlying cause; if it be constitutional, treatment from that standpoint. Some vaginal discharges are due to psychic or neurotic disturbances. Local treatment in the way of douches and tampons may bring about a relief, but long continued treatment of this sort is unsatisfactory. A. C. WILLIAMSON.

Curtis: Bacteriology and Pathology of Fallopian Tubes Removed at Operation. Surgery, Gynecology and Obstetrics, 1921, xxxiii, 621.

Curtis subjected fallopian tubes removed from three hundred patients to a careful and systematic study. Not only were the tubes examined histologically, but, in one hundred and ninety-two of the cases, all material not needed for sectioning was ground up and incubated with suitable culture media. Growths were obtained in thirty-eight cases; of these thirty-three showed macroscopic, and the other five showed microscopic evidence of active inflammation. In nineteen cases the gonococcus was grown, in nine, various forms of streptococci; the colon bacillus was found three times, the bacillus proteus once; three showed mixed growth.

From the clinical history, examination of the external genitalia, and evidence obtained at operation, together with laboratory findings, it was determined that the gonococcus was the offender in 70 per cent of the cases. In a further 10 per cent the gonococcus was thought to have been the primary cause. In about 15 per cent, the tubal pathology was thought to have been caused by the pus-producing bac-

teria. Localized tuberculosis was found in 5 per cent of the cases. It was not possible to obtain growths of gonococcus in any case which had been free from fever and leucocytosis for more than ten days or two weeks. Streptococci, however, were found years after the acute process had subsided.

Gonococcal infection was usually localized in the tube, while other bacteria, which do not have the same tendency to occlude the fimbria, spread to neighboring tissues, but produced less damage within the tubal mucosa. From the material at hand, Curtis concludes that a single attack of gonorrheal salpingitis is borne without protracted clinical symptoms or severe permanent damage, and that grossly thickened tubes are usually the result of repeated infections, either from without or from the lower genital tract. He also found that the adhesions from gonococcal infection are not as firm as those produced by other bacteria.

Since hemorrhage sometimes occurs in thickened gonorrheal tubes, the diagnosis of tubal gestation should be made with caution. Unless carefully looked for, tuberculous salpingitis is easily overlooked. Since tubes infected with gonococci are usually sterile, their removal is not so prone to produce postoperative infection of the ovaries. Tuberculous or septic tubes of like severity, therefore, would call for more radical interference. R. E. WOBUS.

Curtis, A. H.: Problems Concerning Infections of Cervix, Body of Uterus, and Fallopian Tubes. Journal of the American Medical Association, 1923, lxxx, 161.

From a thorough study of one hundred and eighteen uteri, Curtis is able to show that a chronic infection of the endometrium rarely exists, therefore, the uselessness of the curette. He gives two sources of chronic leucorrhea: (1) the urethra, and its surrounding ducts and glands; (2) the cervix. He treats the cervix with radium, and has produced a complete recovery in ninety out of one hundred and four patients. An investigation of diseased fallopian tubes in a series of three hundred cases showed tuberculosis in 5 per cent, streptococcic infection in 10 per cent, and the gonococcus probably responsible for the remainder. In no case was he able to culture the gonococcus in the tubes two weeks after subsidence of fever and leucocytosis. W. KERWIN.

Burns: Chronic Endocervicitis and Its Treatment. Journal of Obstetrics and Gynaecology of the British Empire, 1922, xxix, 619.

Chronic endocervicitis should be considered as a distinct pathological entity entirely apart from endometritis. Discomforting vaginal discharges are usually due to cervical infection. Cultural examination of chronic infected cervices revealed the staphylococcus albus, alone or with other organisms, in 68 per cent, the gonococcus in less than 4 per cent of cases. The entire gland structure is infected in each case. Ionization affords a method of applying treatment throughout the infected area. The author obtained good results by this method except where erosion coexisted. Displacements of the uterus and intrapelvic complications, such as inflammation of the tubes, contraindicate ionization. Removal of the lower two-thirds of the cervical canal is advocated for all cases of chronic endocervicitis accompanied by erosion. H. W. SHUTTER.

Hobbs, Remington: Acute Infections of the Endometrium. British Medical Journal, July 9, 1921, p. 35.

The author advocates careful but active treatment of endometrial infections of whatever character. In cases with gonococcal infection the following points must be carefully observed: All acute cases must be operated on immediately;

the structures below the cervix must be carefully cleaned and made aseptic from below upwards; no attempt should be made to catch hold of, or pull on, the cervix with a volsellum, nor to dilate the cervical canal; and finally, the uterus should be rid of any residual fluid after syringing, and a tube left in for drainage. The author reports several illustrative cases.

For cases of puerperal sepsis he believes in intrauterine treatment with drainage of the uterine cavity. He reports illustrative cases. Conclusions are as follows: (1) Careful treatment of the endometrium with drainage of the uterine cavity lessens the tendency to extension of the inflammation. (2) The uterine cavity may be treated time after time with beneficial effect. (3) Inflammatory conditions of the adnexa are favorably influenced by appropriate treatment of the endometrium. (4) Pain originates very frequently more from inflammation of the uterus than from that of the adnexa. (5) Operations for lesions outside the uterus except those of the grossest character are not indicated until the endometrium has been carefully treated. F. L. ADAIR.

Stajano, C.: Diaphragmatic Reaction in Genital Infection. Gynécologie et Obstétrique, 1922, vi, 44.

Upper abdominal pain is not infrequent in acute pelvic diseases. One should not conclude from upper abdominal symptoms that the lesion is necessarily located in that vicinity. He thinks the diaphragmatic reaction may appear in the following conditions: gonorrheal pelvic peritonitis; menstrual congestion associated with gonorrheal infection; in the early postoperative hours, following liberation of numerous adhesions, especially of acute pelvic adhesions; pelvic lymphangitis; more often in gonorrheal pelvic infections than in pelvic tuberculosis; during the course of peritoneal exudations, but one does not see the reaction in chronic salpingitis, even in the presence of marked lesions. F. L. ADAIR.

Forgue: The Question of Ascending Tuberculous Infection of the Female Genital Tract. Paris Médicale, 1922, xii, 506.

While tuberculous infection of the female genital tract usually occurs by way of the blood stream, or by continuity from the peritoneal cavity, Forgue believes that ascending infection does occur at times. In animals it has been found difficult to produce such infection by way of the blood stream, while comparatively easy by introducing tubercle bacilli into the vagina.

Keogh has shown that tuberculous endometritis is not as rare as is generally supposed, and frequently exists without coexisting infection of the tubes. Forgue believes that, even when the entire genital tract is involved, if the lesions are more advanced in the lower portions, we may assume that the infection ascended from the vagina. He thinks that the infection may ascend by continuity or may even be carried upward by the spermatozoa. R. E. WOBUS.

Linzenmeier: The Rapidity of Sedimentation of Suspended Red Blood Corpuscles as an Aid in Differential Diagnosis in Diseases of the Adnexa. Zentralblatt für Gynäkologie, 1922, xlvi, 535.

Linzenmeier refers to a former article in the above journal, No. 10, 1921, in which he had pointed out the importance of the sedimentation test in the diagnosis of 30 cases of acute or exacerbated infections of the genitalia. Since then Geppert (Berl. klin. Wchnschr., 1921, p. 226), has also discussed this aid in diagnosis of purulent infectious processes, and believes that when the sedimentation

is slow, it is possible to rule out with great certainty acute and subacute processes. Similar satisfaction has been noted by Löhr who states that all inflammations of whatever nature give a sedimentation which is proportional to the severity of the infection, and the extent of the inflammatory processes. He believes that the reaction is of great importance in differentiating inflammatory and noninflammatory bone and joint diseases. Westergreen (Acta. Med. Scand., 1920, liv), claims that the sedimentation reaction is a much more exact measure than temperature after inoculation with tuberculin. "Sedimentation" is a general reaction like fever. If then the corpuscles sink quickly it is not positively certain that the influence is from any localized area, but slow precipitation, on the other hand, allows the elimination of purulent processes anywhere in the body. When done with care, and associated with careful observation of the body as a whole, much information is obtained, particularly in genital inflammatory conditions.

The technic of the test is simple and is described in detail in this article. The method has been used in extrauterine pregnancy and pelvic inflammatory disease in general, and according to Linzenmeier, sedimentation did not occur in less than 40 minutes in any case of extrauterine pregnancy save where there had been an acute intraabdominal bleeding. Here the results were similar to those in acute inflammatory processes. The differential diagnosis between acute peritonitis and severe intraabdominal bleeding is best effected by the estimation of hemoglobin.

With special reference to diseases of the adnexa Linzenmeier brings evidence that in general the slower the sedimentation the better the prognosis for operation.

(a) Where sedimentation occurs in 30 minutes or less it is possible to make a practically certain diagnosis of inflammation rather than extrauterine pregnancy, save in the cases of sudden and acute abdominal bleeding; and (b) in chronic cases the slower the sedimentation the greater the probability of tubal pregnancy. When the sedimentation time is less than an hour, virulent organisms may still be present and operation should, if possible, be postponed, but where it is over two hours laparotomy may be undertaken without fear of latent infection.

LITTLE.

Dalché: Noninflammatory Adnexal Disease. Le Progrès Médical, July 30, 1921, p. 359.

Dalché differentiates between a true inflammatory and a purely congestive condition of the adnexa. In this latter condition the most important etiologic factors are exposure to cold and traumatism, especially if occuring coincident with the menstrual period. Sexual excitement, fatigue, hepatic pathology, or enteric disorders may also cause tubal congestion. In these conditions, the pain is of a paroxysmal nature and often far out of proportion to the apparent pathology. A slight but persistent fever may be present. There is little or no peritoneal involvement and the tubal mass which may sometimes reach the size of a fetal head, is freely movable and separate from the uterus. Menstrual disturbances are a common complication.

Under appropriate treatment the condition may clear up entirely, or, on the other hand, such sequelae as prolapsed ovary, hydrosalpinx, hematosalpinx, or tuboovarian cyst may follow. The treatment advocated by Dalché consists in absolute rest in bed, mild catharsis, small hot or tepid douches under low pressure, hot wet applications to the lower abdomen, and hot baths. Cupping of the anterior abdominal wall may be of value, though it should not be practiced if there is a chance of a laparotomy being necessary. Antipyrine, potassium bro-

mide and laudanum are given as required to relieve pain. During convalescence the patient should be instructed to avoid constipation, and exposure to cold or fatigue. THEODORE W. ADAMS.

Burgess, A. H.: The Diagnosis of Acute Abdominal Crises. British Medical Journal, 1920, No. 3128, p. 877.

The author divides symptoms into general and local. General symptoms include: Shock or collapse, changes in pulse, alterations in temperature, respiration changes, and miscellaneous symptoms such as a dry tongue, hiccough, strangury, and tenesmus. Local symptoms are divided as follows: (1) Abdominal pain. In the early stages this is reflex and not localized to the site of the trouble; later, as a result of the irritation of the parietal peritoneum, the localization is of considerable value. (2) Cutaneous hyperalgesia. These areas have been worked out and described by Mr. David Ligat whose diagrams are reproduced. (3) Localized tenderness is always of value. Localized pressure tenderness arises from either cutaneous hyperalgesia or localized parietal peritoneal irritation. The general abdominal tenderness is of no specific importance unless associated with muscular rigidity. (4) Vomiting when early is a reflex phenomenon, but when recurrent or persistent is always a serious sign. (5) Muscular rigidity. Differentiation of involuntary from voluntary muscular rigidity is important; muscular rigidity due to defective respiratory mobility should be eliminated. (6) Meteorism localized is not necessarily serious. A steadily progressive distension is ominous. (7) Free gas in peritoneal cavity. Positive loss of liver dullness is of great importance. (8) Free fluid in peritoneal cavity. (9) Presence of definite abdominal or pelvic swelling. (10) Rectal examination is very important.

Two fallacies of importance must be kept in mind: The crises of tabes dorsalis, and the apparent abdominal crises resulting from intrathoracic disease. The author groups his cases into (1) the colics, (2) perforations, (3) hemorrhages, (4) inflammations, and (5) obstructions. F. L. ADAIR.

Wilson: Pelvic Inflammation. Medical Journal of South Africa, 1922, xvii, 112.

Pelvic inflammations are classified as follows: (1) puerperal pelvic cellulitis, and (2) pelvic peritonitis, puerperal and nonpuerperal. Pelvic inflammations cannot be classified according to the infecting organisms. Bacteriologically the infections are autogenous in a considerable percentage of cases. Pelvic cellulitis is almost entirely of puerperal origin. The induration begins at the bases of the broad ligaments near the cervix and extends outward along the planes of radiating fibromuscular bands. If the veins are invaded, thrombophlebitis results. Thirty to fifty per cent of the deaths from puerperal sepsis are due to septic thrombophlebitis.

The portal of entry in puerperal pelvic peritonitis is usually at the placental site. Pyosalpingitis is rare after full term delivery while pyosalpingooophoritis is the rule in infections following abortion. The usual lesion in puerperal peritonitis is an abscess of the broad ligament with involvement of the ovary. In sepsis, conveyed along the veins and lymphatics draining the placental site, the appearance of symptoms is delayed.

The vast majority of nonpuerperal pelvic infections are of gonorrheal origin. Complement fixation is of little diagnostic aid in these cases. Surgery is not the treatment for acute gonorrheal peritonitis.

Pelvic cellulitis following labor usually accompanies lesions of the cervix and is in a large percentage of cases preventable at the time of labor. Glycerine

gauze packing and the antiseptic methods developed by the war are frequently of value as measures of treatment. If pus forms, drain. Pus cavities after drainage can be irrigated with antiseptic solutions. When sepsis has occurred from an infection at the placental site the organisms are already past the reach of the curette. In postpartum pyosalpinx, operation should be early before physical depletion and dense adhesions occur. The author still curettes for postabortal sepsis. In pronounced sepsis following abortion in which conservative treatment is of no avail, hysterectomy and salpingooophorectomy are done at the end of two weeks. He has never ligated the veins in septic thrombophlebitis without removal of the uterus. In the chronic stages of peritonitis-salpingooophoritis, conservatism is the rule. If the tubes have become mere pus sacs they must be removed. H. W. SHUTTER.

Nürnberger: Clinical Features and Pathologic Physiology of Conservative Operations upon the Adnexa. Zeitschrift für Geburtshilfe und Gynäkologie, 1922, lxxxiv, 606.

The author reviews the results in 194 patients operated upon for inflammatory disease of the adnexa in 10 years. Posterior colpotomy was the operation in 42 cases, unilateral salpingooophorectomy in 35 cases, bilateral salpingectomy with or without retention of the ovary in 31 cases, defundation in 5 cases, supravaginal amputation in 5 cases and panhysterectomy in 18 cases. Seventy-nine of these cases were followed and reexamined. Six more were heard from by letter. The mortality of the 161 conservative operations was 2.9 per cent; of the 23 hysterectomies, no mortalities.

From a study of this material, the author presents the following conclusions. In all cases of inflammatory adnexal tumors, a systematic conservative treatment is first of all to be instituted. If larger Douglas abscesses develop, these should be emptied and drained. Where the abscess cavities are situated laterally, so that in colpotomy the danger of accidental injuries would be too great, incision and drainage should be undertaken only exceptionally and under special indications (high fever with progressive cachexia, threatening perforation, etc.). If long continued conservative treatment is not successful, laparotomy should be carried out. Only when there is isolated unilateral adnexal disease should one be content with removal of one side. Two-thirds of the cases of unilateral salpingooophorectomy showed a continuation of symptoms. In bilateral pyosalpinx, one may expect results only from radical operation. In young women in whom one wishes to retain the menstrual function, the operation of choice is a defundation with bilateral salpingectomy with the retention of some functionating ovarian tissue. In all other cases, total extirpation should be done. The appendix should always be examined and if diseased should be removed. MARGARET SCHULZE.

Scheid, F.: Adnexal Disease in Childhood and its Significance in the Differential Diagnosis of Appendicitis. Medizinische Klinik, 1922, xviii, 1277.

During the last three years, among 150 women in whom a diagnosis of appendicitis had been made, 30 were found to have salpingitis. The author believes that this proportion is smaller than is usually encountered. He gives the histories of three young girls, 15, 13 and 13 years old respectively, in whom a diagnosis of appendicitis had been made, but in whom pelvic disturbances were found, which caused the symptoms demanding operation. In two of the patients the hymen had been intact and menstruation had not occurred. In two cases gonorrheal salpingitis was found, while in the third there was a 360 degrees torsion of

the right tube and ovary. From the occurrence of these three cases in a relatively short period of time, the author believes that adnexal disease in children is not uncommon. If at operation the appendix appears normal, the adnexa should be examined. The diagnosis is difficult. When in doubt, an exploratory laparotomy will do little harm and will be less dangerous than neglecting an appendicitis. J. P. GREENHILL.

Benthin, W.: Obstetrical and Gynecological Free Peritonitis. Monatsschrift für Geburtshilfe und Gynäkologie, 1922, lx, 171.

A critical review of the literature on the use of ether for free peritonitis is presented. In addition, Benthin gives his own results. Of his 71 cases of peritonitis, 47 were obstetrical. Most of the latter were due to abortion, especially of criminal origin. The majority of the gynecological cases occurred after operation. The general mortality for the 71 cases was 50.5 per cent; but omitting those patients (22) who had general sepsis, the percentage of cures was as follows: Of the 49 remaining patients, 71 per cent; the gynecologic cases 63 per cent; those with peritonitis occurring after labor 85.7 per cent; and those with peritonitis after abortion 73.8 per cent.

The author advises that an early diagnosis be made by means of abdominal puncture which is without danger. Operation should be performed even when the diagnosis is only a suspicion. The operation should be limited essentially to incision and drainage, without irrigation or mechanical removal of pus, and without release of adhesions. Before closing the peritoneal cavity, 200 gm. of ether are injected. Of great importance is the aftercare which should include regulation of the fluid intake, and care of the heart and bowels. J. P. GREENHILL.

Benthin, W.: Is it Possible to Improve the Prognosis of Obstetrical and Gynecological Peritonitis? Medizinische Klinik, 1922, xviii, 1453.

The prognosis for peritonitis of obstetric and gynecologic origin is much less favorable than it is for surgical peritonitis (70 to 80 per cent mortality against 30 to 40 per cent). The author believes however that it is possible to lessen the mortality in the former group by an early diagnosis with abdominal puncture. If leucocytic exudate is found, operation is done immediately under lumbar anesthesia. Before closing 200 c.c. of ether are poured into the peritoneal cavity and one quart of salt solution containing adrenalin is given subcutaneously immediately after operation, also camphor, caffeine and digalen.

Also in doubtful cases operation should be performed, but should be as conservative as possible. The adnexa should not be removed, and the uterus only in case of perforation, laceration and hemorrhage. Prophylactic enterostomy need not be done, for lumbar anesthesia and ether have a very beneficial effect on the paralyzed intestines. A general anesthetic should be avoided. Morphia may be used freely during the convalescence. J. P. GREENHILL.

Adami: Fulminant Menstrual Peritonitis. Journal of Obstetrics and Gynaecology of the British Empire, 1922, xxix, 104.

The author gives the history and autopsy findings in a case of septic peritonitis occurring in a menstruating 14 year old girl. Less than 24 hours before, while menstruating she had taken part in a gymnasium contest. Death followed a toxic, comatose condition of but a few hours' duration. Autopsy revealed free fluid in the peritoneal cavity and a moderate congestion, most pronounced in the pelvis.

The uterus contained a small amount of sanguinous fluid, the cervix was open and contained no mucous plug. The examination of all other organs was negative. A pure culture of streptococcus was obtained from the free peritoneal fluid. So sudden and profound had been the attack that no leucocytic reaction occurred.

Adami concludes that the negative intraabdominal pressure produced repeatedly while swinging on gymnasium rings had aspirated vaginal contents, through the open cervix into the abdominal cavity. A somewhat similar phenomenon may account for some of the idiopathic puerperal infections seen in practice.

H. W. SHUTTER.

Block, F. B., and Mikelberg, H. M.: The Nonoperative Treatment of Pelvic Inflammatory Disease. Pennsylvania Medical Journal, 1922, xxvi, 15.

One hundred and twenty cases of chronic inflammatory pelvic disease received nonoperative treatment, consisting in hot douches at home, local treatment of cervical infections, application of antiphlogistic solutions, and in some cases administration of organic glandular preparations. Of 91 patients with gross pathology, 19 were much improved, 49 showed only moderate improvement, a total of 68 (or 74.6 per cent) showing improvement. The remaining 23 patients (25.4 per cent) were unimproved, and 5 of these were referred for operation. Of the 29 patients without gross pathology, 8 were much improved, 12 moderately improved, a total of 20 (68.9 per cent) showing improvement; while the remaining 9 patients (31.1 per cent) there was no improvement.

In brief, 98 cases (81.6 per cent) were treated less than four months, and were either improved sufficiently to discontinue treatment, or were advised to seek operative relief.

C. O. MALAND.

Campbell, John: Treatment of Salpingooophoritis. British Medical Journal, October 14, 1922, p. 683.

The author deals with the treatment under two separate headings.

(1) Acute salpingooophoritis. Irrespective of the cause, the prevailing opinion is in favor of expectant treatment during acute inflammatory condition of the tubes and ovaries. The author is of the opinion that acutely inflamed tubes should be removed as early as possible.

(2) Chronic salpingooophoritis. He divides his cases into two groups; first, the patient in whom symptoms are practically absent, the woman usually seeking advice on account of sterility; second, the patient whose general health is poor with much pain and discomfort in the pelvic region. In the first group expectant and local treatment has a place. In the second group, operation is indicated except in syphilitic cases. The author prefers the abdominal route in his operations. He feels that drainage is necessary in certain cases, preferably using strips of gauze sheathed with thin rubber.

F. L. ADIAR.

Dubose: Treatment of Acute Pelvic Infections in Women. Surgery, Gynecology and Obstetrics, 1921, xxxiii, 299.

Instead of awaiting developments, Dubose advises immediate operation in acute pelvic inflammation, preferably within the first twelve hours. He makes a low abdominal incision and removes all acutely infected organs as well as any pyogenic membranes present, even in cases where there is a distinct fluctuating mass in the culdesac. If seen after twelve hours, he sometimes defers operation in cases where the process seems stationary, or if a high leucocyte count demon-

strates active resistance to the infection. According to this plan, he has operated on 255 cases, including 80 hysterectomies, with one death. There were 76 double salpingectomies in addition. In the rest he removed either one tube or ovary or both.

While Dubose claims complete cure in the majority of his cases, he admits that not one subsequent pregnancy occurred in any of these patients.

R. E. Wobus.

Werner, P.: On the Treatment of Genital and Peritoneal Tuberculosis. Wiener Klinische Wochenschrift, 1922, xxxv, 535.

Operative treatment has been used by French surgeons while conservative treatment has been advocated by the Germans (especially Amann, Martin, Veit, and Krönig.) The difficulty of making an accurate diagnosis without operation makes the statistics in regard to cure by medical methods uncertain. In making a diagnosis the past history is important. Late appearance of menstruation with dysmenorrhea is in favor of genital tuberculosis; abnormal menstruation is of little value. Fever with tumor is in favor of a positive diagnosis. Pain varies greatly in degree and situation. Palpation is not reliable, though on the whole there is less spasm with tuberculosis than with infections. The eye, skin, and tubercular tests are not certain proofs as there may be tuberculosis elsewhere in the body. The discovery of tubercle bacilli in the uterine discharge is difficult.

The author reports 14 cases of peritoneal and 23 of genital tuberculosis. The classification rests on the dominant type, both being found in most cases. Most of them were treated with x-ray after operation. Of the former 14 cases, 11 were of the ascitic form and 3 of the adhesive; all had exploratory laparotomies. Of the 23 cases of genital tuberculosis 10 had abdominal hysterectomy with the removal of both adnexa. Three had partial removal of the adnexa by laparotomy, two by vagina; one had a large abscess opened, resulting in a recto-vaginal fistula. Four had exploratory laparotomies but conditions were so bad that nothing was removed. One was explored by the vaginal route but such dense adhesions were found that nothing was done.

In the 37 cases he had two operative deaths, 5.4 per cent. There were three fistulae, 8 per cent (two of them in one patient). The end results were hard to verify because of difficulty in following the patients, but over 50 per cent were cured.

X-ray treatment is giving good results with weak dosage so that amenorrhea and sterility do not have to occur.

Frank A. Pemberton.

Jaegerroos: Hydrosalpinx. Archiv. für Gynäkologie, 1921, cxiv, 328.

Jaegerroos offers an exhaustive clinical and histologic study of hydrosalpinx, based on 100 cases operated upon, 6 in Schauta's clinic and 94 in Engstroem's. The chemistry of the fluid in 14 cases studied, and the histology of the tubes in the 58 cases studied convince the writer that the hydrosalpinx begins as a salpingitis in which the exudate is either serous or slightly purulent, with a transudate following. Other studies of his own have convinced him that this salpingitis is limited in cause to one of three organisms: streptococcus (puerperal), tubercle bacillus, or commonly gonococcus.

The dilatation of the tube is usually in the ampulla, and the maximum size observed was that on a fetal head. Because of its bearing on operative treatment, it is worthy of mention that Opitz has explained the invariable inrolling

of the fimbriated end of the tube on the grounds that the tube, elsewhere loosely invested by peritoneum, has a snug peritonal collar close to the abdominal ostium: an accumulation of fluid medial to this collar distends the tube and causes the fimbriated end to roll through the constriction. In favorable cases, therefore, evacuation of the fluid makes possible a restoration of the fimbriae to their normal position. A certain optimism as to restoration of function is justified by Jaegerroos' always finding the epithelium preserved, though much flattened where the tube is markedly distended. In several cases the fresh specimen showed active motion of the cilia. Based on the favorable results of salpingotomy or salpingostomy, or both, in Engstroem's clinic, Jaegerroos pleads for conservatism where it is possible. In 59 cases so treated which were followed up, there were only 5 found with swellings in the adnexa, after from 9 months to 7 years. One patient bore a living child 18 months after salpingostomy.

RAMSAY SPILLMAN.

Nash: Hemotosalpinx-Pyosalpinx with Torsion of the Right Fallopian Tube. The Lancet, 1922, ccii, 78.

This condition occurred in an unmarried girl 18 years of age. The interest in this case lies in the torsion of a tube already the seat of pyosalpinx, and in the resemblance of the symptoms to appendicitis. It illustrates the need of investigating the condition of the pelvic organs when there is insufficient evidence of disease in the appendix.

NORMAL F. MILLER.

Books Received

PHYSIOLOGIE OBSTETRICALE, NORMALE ET PATHOLOGIQUE. Par H. Vignes, Accoucheur des Hôspitaux de Paris. Préface du Professeur A. Couvelaire. Masson et Cie, Éditeurs, Paris, 1923.

OBSTETRICS FOR NURSES. By Charles B. Reed, M.D., Obstetrician to Wesley Memorial Hospital, Chicago. Second edition, with 144 illustrations, including two color plates. C. V. Mosby Company, St. Louis, 1923.

TRAITE D'OBSTETRIQUE. Par A. Ribemont-Dessaignes et G. Lepage. Neuvième Édition, revue et mise à jour par V. Le Lorier. Avec 587 figures dans le texte. Masson et Cie, Éditeurs. Paris, 1923.

AIDS TO GYNAECOLOGY. By Richard E. Tottenham, B.A., M.D., etc. Censor and Examiner in Midwifery; Fellow of the Royal Academy of Medicine, Ireland, etc. Sixth Edition. William Wood & Company, New York, 1923.

HEREDITY AND CHILD CULTURE. By Henry Dwight Chapin, President of the Children's Welfare Federation of New York, etc. With a Foreword by Professor Henry Fairfield Osborn. E. P. Dutton & Co. New York, 1923.

BIOLOGIE UND PATHOLOGIE DES WEIBES. Ein Handbuch der Frauenheilkunde und Geburtshilfe. Herausgegeben von Josef Halban, Wien, und Ludwig Seitz, Frankfurt a.M. Lieferung 1 (III. Band, Seiten 1-464, mit 171 Abbildungen im Text). Verlag von Urban und Schwarzenberg, Berlin und Wien, 1923.

HANDBUCH DER KINDERHEILKUNDE. Ein Buch für den praktischen Arzt. Herausgegeben von Professor Dr. M. von Pfaundler und Professor Dr. A. Schlossmann. Erster Band, dritte Auflage. Verlag von F.C.W. Vogel in Leipzig, 1923.

Erratum

In Dr. Thos. R. Goethals' article in the September issue, page 329, Table IV, the last figure in the last column should read 2.56% instead of 0.64.